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A CONGENITAL ANOMALY OF THE FUNDUS OCULI

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THE HAGUE, THE NETHERLANDS

REPORT OF A CASE

De B., a soldier 19 years of age, was sent to the ophthalmic department of the military hospital in The Hague because of decreased vision in the left eye, which could not be corrected by glasses. The young man did not remember whether he had ever been able to see well with this eye.

The left eye deviated to a slightly divergent position. The vision was nearly ¼ (Snellen); there was no improvement with lenses. Skiascopy without cycloplegia revealed apparent low hyperopic refraction, which later could not be verified. The pupillary reactions were intact. In the anterior segment of the eye no abnormalities could be seen with the slit lamp. There were no evidences of past inflammation or signs of a persistent pupillary membrane. A little pointlike opacity on the posterior surface of the lens, somewhat to the nasal side and below the center, possibly marked the place of attachment of the hyaloid artery; there were no remains of this vessel in the vitreous. On slit lamp examination with the pupil dilated, the vitreous was found to be of normal structure and transparency. The ophthalmoscopic picture of the left eye is shown in figures 1 and 2.

A cudgel-like white mass lay with its central end in front of the temporal edge of the papilla nervi optici; it ran upward toward the temporal side. The sharply outlined central swelling of the cudgel lay at a level of +2 D., whereas the vessels emerging from the papilla, at the point where they passed onto the retina, were at a level of —1 D. This degree of prominence of the white mass in the vitreous remained about the same up to the peripheral knoblike end, which was situated just past the fovea centralis. In the central swelling of the cudgel-like mass a groove was visible. This groove ran in the direction of the cudgel and became deeper as it passed from its peripheral beginning to the center. It continued more centrally in the form of a tube which penetrated in a slanting direction and seemed to end in the papilla nervi optici at the point where the retinal vessels emerged. The posterior side of the cudgel-like mass was adherent to the retina along its whole length and caused the latter to be lifted up from its pigment layer like a tent.

A retinal artery was divided into many branches, which ran along the anterior upward-slanting surface of this localized detachment. At the beginning of their course the arteries showed many windings both along the surface of the retina and perpendicularly to it. The arteries and veins could be easily distinguished from each other.

The white, semitransparent cudgel-shaped formation was finely striped, the stripes running in the direction of the groove. The same tissue was continued as a

OPK

From the Army Hospital.

thin, transparent veil from the main mass downward to the temporal side and to the nasal side of the papilla. It was limited below by a sharp line in the form of a horizontal, elongated S, lying freely in the vitreous. At both ends of the cudgel-like mass this veil formation radiated toward the retina, covering centrally also the nasal part of the papilla, which could be seen through it. Some thread-like shoots extended from the dense white mass into the vitreous, where they branched and ended freely. The definite white thread, which ran upward from the central swelling, could be followed to a distance of + 5 D. in the vitreous.

So far as I could see, in reference to its transparency, the preretinal white tissue did not contain any blood vessels. The pigmentation of the fundus oculi was the same as that of the right fundus. The pattern of the choroid could hardly be seen. The pigmentation of the retina was evenly distributed, except that the floor of the tentlike retinal detachment was darker than could be attributed simply to shadow influence. The visual field of the left eye showed an absolute scotoma as large as the blindspot, as well as a few central indistinct scotomas difficult to outline accurately. The absolute scotoma lay about as far to the nasal side as the blindspot lies to the temporal side of the point of fixation, but also somewhat lower. This scotoma was evidently due to the peripheral knob of the cudgel-shaped formation.

The right eye was myopic (— 0.75 D), but otherwise normal.

Fig. 2.—Ophthalmoscopic picture of the left eye of a patient with ablatio falciformis congenita.

COMMENT

Without confirmation by pathologic examination, the following explanation for this congenital anomaly may be given: The white mass is a hyperplasia of the fetal neuroglia (Bergmeister's papilla). This glial tissue reaches its maximal extent in the fifth month of fetal life, forming a system of funnel-shaped sheaths around the fetal hyaloid vessels, which afterward disappear. Small remnants of this glial tissue on the papilla often lead to a diagnosis such as that of glial veil or membrana epipapillaris.

According to my former hypothesis [1] for the origin of the ablatio falciformis congenita [2] (retinal fold [3]), one must consider the cause in this case also as a fetal adhesion between the wall of the peripheral

1. Van Manen, J. G.: Arch. d'opht., to be published.
2. Weve, H.: Arch. f. Augenh. **109**:371, 1935.
3. Mann, I.: Brit. J. Ophth. **19**:641, 1935.

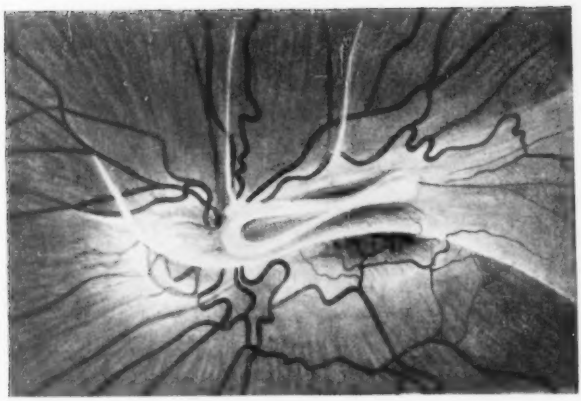

Fig. 1.—Ophthalmoscopic picture of the left eye of a patient with ablatio falciformis congenita (drawn by the artist of the Netherlands Hospital for Ophthalmic Diseases, Utrecht).

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hyaloid vessel (one of the vasa hyaloidea propria) and the inner layer of the retina. When the eye grows and the primary vitreous is pushed to the center, this abnormally persisting tissue is tightened and runs like a cord through the eyeball. The nerve layer of the optic cup is prevented by this adhesion from following the pigment layer along the inner side of the eyeball, and so a tentlike ablatio retinae is formed.

The groove in the central swelling of the cudgel-like mass of white tissue is the entrance to the tunnel through which the main branch of the hyaloid artery has run. It is remarkable that the hyaloid artery itself disappears in a normal way, so that there are no remains of its distal end to be found on the posterior surface of the lens.

An anomaly of the fundus described by Danis [4] evidently belongs to the same group of conditions as the one just described. Danis did, however, find the remains of the central hyaloid artery, both its proximal origin on the papilla and its distal end on the posterior surface of the lens. In the colored illustration in Danis' article, a blue-white cord protrudes into the vitreous from the peripheral end of the prominent white mass on the papilla and is correctly described as a persisting hyaloid artery. But I consider the bandlike white mass which passes back to the retina more peripherally to be a foldlike retinal detachment, a view which Danis did not take. The pigmented stripes along both longitudinal edges of this white mass bear out my statement. The same pigmented border is seen in cases of ablatio falciformis congenita.

In the 24 year old man described by Danis, the head of the optic nerve had come to lie in the eyeball, the nerve having been drawn in the direction of the adhesion which hampered its growth (persistence of the intercalated neuron of von Szily [5]). Hence, the abnormal distribution of retinal vessels, as in this case, which grow out from the papilla in a later stage, and the pigmented edge on the side of the papilla, which is opposite to the side of traction, are regularly observed in cases of ablatio falciformis congenita.

In typical ablatio falciformis congenita the retinal fold is continued from the papilla nervi optici right up to the periphery; the fold lies in the temporal meridian, just as the partial foldlike ablatio does in the case described.

In the first of the 2 cases of rare congenital anomalies of the fundus published by Botteri,[6] the retinal fold ran nasalward. This case was very similar to that of Danis, judging from the illustration, but the point of attachment to the lens of the hyaloid artery after it left the white mass could not be determined. In Botteri's second case the band of

4. Danis, M.: Am. J. Ophth. **4**:233, 1921.

5. "Schaltstück von von Szily."

6. Botteri, A.: Klin. Monatsbl. f. Augenh. **48**:11, 1910.

blue-white tissue which ran from the papilla beneath the macula to the temporal side could be followed through the vitreous to the equator of the lens. In this case many pigmented spots distributed over the periphery of the retina were described.

In the illustration of the case reported by Finnoff [7] the retinal fold runs upward in the vertical meridian almost to the periphery. A shining green cord can be seen at its origin on the papilla, somewhat in front of the white mass, but near the periphery of the mass it disappears. I consider this cord also as a peripheral branch of the hyaloid artery (vasa hyaloidea propria) and the source of the malformation.

I also observed a case of the same condition in the Netherland East Indies and described it in a report.

The condition in all the cases described and cited in this paper was unilateral. Ablatio falciformis congenita is as a rule bilateral, although the degree of the malformation is usually not the same on the two sides.

Weve [8] showed that ablatio falciformis congenita in its varying degrees has a tendency toward familial occurrence. My study of the literature on this condition and related anomalies has shown that often among the relatives of persons affected by these congenital anomalies other congenital ocular conditions are found.

The father (60 years old) of my patient had not been able to see well from his early youth. On examination his right eye had a vision of ¼ and his left eye of ½ (Snellen). The refraction determined by glasses showed on both sides 0.75 D., or hyperopia; the vision, however, was not improved by the glasses.

Examination with the slit lamp (with dilated pupils) revealed symmetric congenital opacities of the lens in both eyes. These opacities consisted in a definite anterior and a slight posterior suture cataract, irregular spider-web-like opacities centrally situated in the lens in front of the anterior Y-shaped cataract, innumerable points and small striped gray-white opacities situated peripherally to the clear nucleus and increasing in size toward the periphery and a beamlike opacity in the periphery of the lower nasal quadrant. The point of attachment of the central hyaloid artery to the posterior surface of the lens could not be seen.

An ophthalmoscopic examination showed that the fundus was normal, apart from a few white spots in the neighborhood of the fovea, which I considered to be "drusen" (as well as I could judge from looking through the central lens opacity). The papillae nervi optici were normal. At the periphery there were small round and fine wreathlike pigmentations and depigmentations. The visual fields showed a slight concentric limitation. Adaptation was not determined, but there were no complaints of night blindness. On the contrary, the man, who worked as an artist indoors, complained that his sight was much worse in the brighter light outdoors. This was understandable because of the location of the lens opacities.

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7. Finnoff, U. C.: Am. J. Ophth. **3**:161, 1920.
8. Weve, H.: Brit. J. Ophth. **22**:456, 1938.

Weve described and illustrated pigmentation in the fundus similar to that described here in a family two members of which had an atypical unilateral rudimentary retinal fold. The pigmentation in his cases extended more toward the center and resulted in night blindness and considerable concentric contraction of the fields.

Hoffmann [9] described an abnormal distribution of pigment in the periphery of the fundus in two brothers of a patient with typical ablatio falciformis congenita.

The only brother of my patient, 16 years old, had no ocular abnormality.

The examination of the father is interesting because the congenital opacities of the lens can be considered the result of a fetal disturbance in the nutrition of the lens through the hyaloid vessel system.

Hence, it might be concluded that the same disturbance in the germ of the father and of the son gave rise to two different congenital abnormalities of the eye.

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9. Hoffmann: Klin. Monatsbl. f. Augenh. **77**:370, 1926.

DEEP CUPPING OF NERVE HEAD IN ATROPHY OF OPTIC NERVE DUE TO METHYL ALCOHOL

COMPLICATED BY AMBLYOPIA DUE TO TOBACCO AND ETHYL ALCOHOL

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A few of the patients who survive methyl alcohol poisoning subsequently have a type of atrophy of the optic nerve which is accompanied with deep cupping of the disk. The fundus picture is so similar to that associated with glaucoma that when the condition is encountered in practice a great deal of proof is required before deciding against a diagnosis of glaucoma. Fridenberg [1] first described this picture as an entity in 1910, although Moulton [2] mentioned cupping in 1 of his cases in 1901. In the acute stage the disk appears intensely red and is definitely blurred, with extension of the edema to the peripapillary portion of the retina. The swelling increases in prominence; the color of the disk fades into white. Full and permanent atrophy rapidly follows, featured at times by the remarkable excavation of the nerve head which is so puzzling. No elevation of tension is present. Additional cases were reported by Brown,[3] de Schweinitz [4] and Wilder [5] in the discussion of Fridenberg's paper. In the same year Gruening [6] published a description of a similar case, with illustrations of the disks drawn by Fridenberg. There were excavations on both sides, 6 D. in depth. The right disk contained pigmentary traces of a hemorrhage. Cupping has been observed four to four and one-half months after the poisoning.[7]

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1. Fridenberg, P.: Tr. Am. Ophth. Soc. **12**:513, 1910.

2. Moulton, H.: A Case of Blindness from Drinking Bay Rum, Compared with the Reported Cases Due to Methyl Alcohol and to Essence of Jamaica Ginger, etc., J. A. M. A. **37**:1447 (Nov. 30) 1901.

3. Brown, E., in discussion on Fridenberg.[1]

4. de Schweinitz, G. E., in discussion on Fridenberg.[1]

5. Wilder, W., in discussion on Fridenberg.[1]

6. Gruening, E.: Arch. Ophth. **39**:333, 1910.

7. Shannon: A Case of Bilateral Optic Atrophy Due to Methyl Alcohol Poisoning, Arch. Ophth. **7**:813 (May) 1932. Morrison, F.: Am. J. Ophth. **5**:645, 1922.

REPORT OF A CASE

Mr. G. H., aged 46, a marine engineer, presented himself on Jan. 15, 1940, stating that his reading glasses were vaguely unsatisfactory. He wore for the right eye + 0.75 and for the left + 1.50 sphere, with addition for both eyes of + 1.50 sphere for reading, given to him by an optometrist five months previously. He admitted that in 1918 he had suffered from wood alcohol poisoning and that vision in the right eye had been defective since that time. About 1935 an oculist made the diagnosis of glaucoma in both eyes, but in 1937 another oculist informed him that no elevation of tension existed. The patient was in the habit of smoking forty cigarets daily; he consumed about a quart (950 cc.) of liquor per week and about six cups of coffee daily, and his routine of eating and sleeping was extremely irregular.

The vision in the right eye with a + 0.75 sphere was 20/50, and in the left eye with a + 1.25 sphere, 20/25 —. The pupils were normal in their reactions but were dilated 6 mm. Examination of the right fundus revealed marked cupping of the nerve, about 4 D. in depth, extending to the very margins. Well established greenish gray atrophy was present. The markings of the lamina cribrosa were not evident. The arteries were much reduced in caliber. A narrow irregular margin of chorioretinal atrophy surrounded the disk. The left disk was somewhat less deeply excavated and was grayish. The lamina cribrosa was obscured and the arteries were thin. A large number of fine and coarse pigment clumps were scattered in the hollow of the cup, a replica of the hemorrhage remnants described by Gruening. The vessels in both disks were crowded nasally.

The tension with the McLean tonometer was 28 mm. of mercury in the right eye and 30 mm. in the left. After an hour in the dark the tension did not rise. Wide dilation of the pupils did not induce an increase in intraocular pressure. The nocturnal and diurnal tension curves were normal.

The fields were rather bizarre and did not tally with the picture of glaucoma. The right field (3 mm. white test object; distance, 1 meter) showed constriction to about the 25 degree circle except nasally, where it fell just within the 20 degree boundary. The blindspot was moderately enlarged, and from its upper pole extended an arcuate scotoma along the 10 degree circle to a distance slightly above and beyond the fixation point. No central defect was demonstrable, but a large circular scotoma was found 10 degrees below the fixation point.

The left field was reduced to a sinuous remnant shaped like a reversed interrogation mark. There was a relative central scotoma about 2 degrees in diameter. The location of the blindspot did not fall within the seeing area.

Roentgen studies of the skull, sinuses and optic canals gave negative results. Physical and neurologic examinations also revealed no abnormalities. The Wassermann reaction was negative.

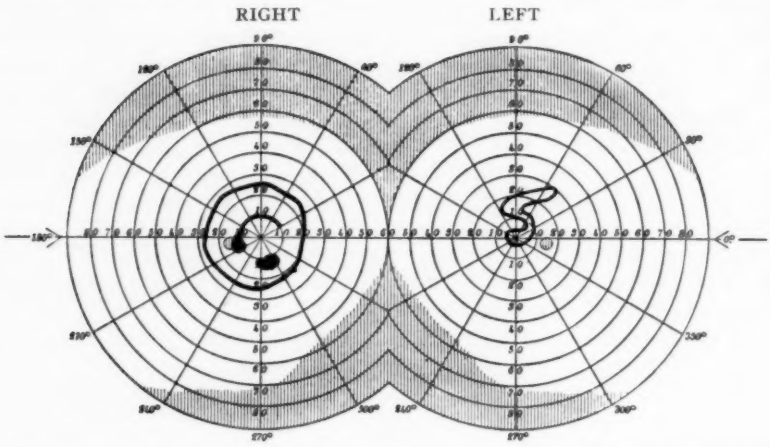
I was satisfied that glaucoma could be excluded, and in the absence of other pathologic observations I felt convinced that the cupping was of the type caused by methyl alcohol. I increased the power of his reading glasses and informed him that no treatment was indicated.

In March, however, the patient returned with the complaint that his new glasses were not appreciably better than the old ones. Objects appeared to him as though viewed "through a smoked glass." This was especially annoying in reading and working. The complaint bore reference to the left eye only, since the right was of little use for fine visual discrimination. The corrected vision was 20/50

in the right eye and 20/30 + in the left. No change in the fields was noted. I could find no evidence that the atrophy of the optic nerve was reactivated to progression after so many years, and I felt justified in reassuring the patient that his sight was not in danger.

Four months later the patient broke his glasses and came back for a reexamination. He complained more than ever of foggy vision in the left eye and was extremely worried. His work carried him into dimly lighted engine rooms and holds aboard ship, and he found that inspection of machinery and charts in these places was becoming increasingly arduous. In general, vision at night was noticeably impaired. The vision in the right eye was 20/50 —; in the left eye it had dropped to 20/40 +. The patient now had difficulty in maintaining fixation with the left eye on a very small object. The tangent screen revealed a small relative central defect communicating with the arcuate scotoma previously noted.

The probability of toxic amblyopia due to ethyl alcohol and tobacco superimposed on the old atrophy of the optic nerve belatedly suggested itself;



Visual fields in a case of atrophy of the optic nerve due to methyl alcohol and complicated by amblyopia due to tobacco and ethyl alcohol. Vision in the left eye was 20/25; in the right eye, 20/50. (Tangent screen; 3 mm. test object; distance, 1 meter.)

apparently the premonitory complaints had been the unrecognized heralds. The patient was immediately ordered to abstain from liquor and tobacco, and I prescribed prostigmine bromide (0.0075 Gm.) three times a day. Brewers' yeast was also ordered, and the patient received daily subcutaneous injections of acetylcholine bromide. He was advised to pay more attention to the regularity and quality of his meals. A marked improvement was noted subjectively on the second day of treatment, and within ten days the vision had risen to 20/30 +. This therapy was carried on for about one month and then tapered off. The last examination, on October 30, disclosed the vision to be 20/50 in the right eye and 20/25 — in the left. The patient states that his sight is now as good as it ever has been since 1918. The central scotoma has disappeared from the left field, but the arcuate scotoma remains. Night vision is no longer defective.

Methyl alcohol has no specific tissue affinity, but it injures most severely the highly differentiated neural tissues, particularly the ganglion cells of the retina. It causes parenchymal degeneration of the viscera and exerts a toxic influence on the blood vessels. These facts have been amply verified by animal experimentation [8] and by examination of human autopsy material.[9] The vagus nerves, the phrenic nerves [9b] and the nerves of the upper extremities [10] have been found to suffer. Methyl alcohol is distributed to the tissues in proportion to their water content; all the cells are exposed to practically the same methyl alcohol–water concentration.[9i]

The high toxicity of methyl alcohol is thought to be due to intermediate products which result from its slow and incomplete oxidation. These are formic acid and possibly formaldehyde, the former being six times, and the latter thirty times, more toxic than methyl alcohol.[11] Amyl alcohol and furfural appear not to be present in the adulterated liquor in sufficient concentration to exert much damage.[11] Formic acid is readily recovered from the urine,[12] but free formaldehyde cannot be detected.[13] Formic acid has the property of reducing Fehling's solution; hence the possibility of a false implication of diabetes.

The earlier investigators failed to differentiate the effects of methyl alcohol from those of ethyl alcohol, partly because of conflicting animal

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8. (*a*) Holden, W.: Arch. Ophth. **28**:637, 1899. (*b*) Birch-Hirschfeld, A.: Arch. f. Ophth. **52**:358, 1901; **54**:68, 1902; Ztschr. f. Augenh. **35**:1, 1916. (*c*) Friedenwald, H.: The Newer Pathology of the Retina, with Special Reference to the Changes Produced in the Ganglion Cells by Certain Toxic Agents, J. A. M. A. **37**:1443 (Nov. 30) 1901; Bull. Johns Hopkins Hosp. **13**:52, 1902. (*d*) Tyson, H., and Schoenberg, M.: Experimental Researches in Methyl Alcohol Inhalation, J. A. M. A. **63**:915 (Sept. 12) 1914. (*e*) Scott, E.; Helz, M., and McCord, C.: Am. J. Clin. Path. **3**:311, 1933.

9. (*a*) Bielschowsky, M.: Neurol. Zentralbl. **31**:394, 1912. (*b*) Gettler, A., and St. George, A.: Wood Alcohol Poisoning, J. A. M. A. **70**:145 (Jan. 19) 1918. (*c*) Norris, C.: New York M. J. **3**:583, 1920. (*d*) Eleonskaya, cited by Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, p. 3023. (*e*) MacDonald, A.: XIII Internat. Ophth. Cong., Amsterdam, 1929, p. 440. (*f*) Uher, V.: Voj. zdravot. listy **5**:120, 1929. (*g*) Burhans, E.: Illinois M. J. **57**:260, 1930. (*h*) Jansen, J.: Acta path. et microbiol. Scandinav., 1936, supp. 26, p. 146. (*i*) Yant, W., and Schrenk, H.: J. Indust. Hyg. & Toxicol. **19**:337, 1937. (*j*) Menne, F.: Acute Methyl Alcohol Poisoning: Report of Twenty-Two Instances with Postmortem Examinations, Arch. Path. **26**:77 (July) 1938.

10. Jelliffe, S. E.: M. News **86**:387, 1905.

11. Hunt, R.: Bull. Johns Hopkins Hosp. **13**:213, 1902.

12. Pohl, J.: Arch. f. exper. Path. u. Pharmakol. **31**:281, 1892-1893.

13. Scott and others.[8e] Gettler and St. George.[9b]

experiments [14] and partly because the lack of stringency in government control led to frequently unrecognized adulteration of liquors with wood alcohol. It seems clear at present that the clinical and microscopic manifestations of methyl alcohol poisoning are quite characteristic and should entitle the drug to special classification. As small an amount as 7 to 10 cc. may provoke symptoms; on the other hand, astonishingly large quantities may be imbibed without ill effect,[15] which indicates a probable idiosyncrasy on the part of some of the victims and a high tolerance in others. The loss of vision is usually evident in most cases within twenty-four hours; about the third week the sight begins to recover, and after several weeks the improvement reaches its maximum. At this point the restored vision may be retained, but in most instances it fails rapidly again. The process is bilateral but not necessarily equal in intensity on the two sides. Positive central scotomas (Weekers' sign) appear during the period of temporary recovery.[16]

Histologic examination of affected animal and human eyes has shown that the early lesions may be present only in the retina,[17] from which it follows that the subsequent atrophy of the optic nerve is of the secondary, ascending type. On the other hand, the atrophy of the optic nerve may be primary, with no lesions in the retina, as in the patient examined by Uher eleven years after poisoning,[9f] or, finally, the lesions in the retina and the nerve may be simultaneous, independent and primary to each.[18] The last-mentioned possibilities are more consistent with ophthalmoscopic observations. The retinal lesions are patchy,[8e] which possibly explains the scattered multiple scotomas observed by Tyson [19] in cases of chronic involvement. Ziegler [20] expressed the opinion that the pituitary is the site of the important primary lesions due to wood alcohol poisoning. The action of light has been considered

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14. Igersheimer, J., and Verzar, F.: Arch. f. Augenh. **75**:27, 1913. de Schweinitz, G.: The Toxic Amblyopias, Philadelphia, Lea Brothers & Company, 1896, p. 51. Friedenwald.[8c]

15. Sourdille, G., and Sourdille, G. P., in Bailliart, P.; Coutela, C.; Redslob, E., and Velter, E.: Traité d'ophtalmologie, Paris, Masson & Cie, 1939, vol. 6, p. 807. Cutler, C.: New York M. J. **3**:585, 1920.

16. Weekers, L.: Arch. d'opht. **49**:485, 1932; **51**:497, 1934. Goldflam, S.: Klin. Monatsbl. f. Augenh. **64**:684, 1920. Joiris, N.: Arch. d'opht. **52**:578, 1935.

17. Henderson, Y., and Haggard, H.: Noxious Gases and the Principles of Respiration Influencing Their Action, New York, The Chemical Catalog Company, 1927. Holden.[8a] Birch-Hirschfeld.[8b] Tyson and Schoenberg.[8d] Scott and others.[8e] MacDonald.[9e] Menne.[9j]

18. Friedenwald, J. S., and Felty, A., in Friedenwald, J. S.: The Pathology of the Eye, New York, The Macmillan Company, 1929, p. 124. Bielschowsky.[9a]

19. Tyson, H.: Arch. Ophth. **41**:458, 1912.

20. Ziegler, S.: The Ocular Menace of Wood Alcohol Poisoning, J. A. M. A. **7**:1160 (Oct. 1) 1921.

harmful in cases of methyl alcohol poisoning, an idea reflected in the injunctions of the older clinicians to confine the patients to dark quarters.

In evaluating the reliability of histologic reports great emphasis must be placed on the importance of special staining methods, since mild changes in the nerves and ganglion cells might easily be missed if these were not employed.

To return to the case reported here, there can be no doubt as to the nonglaucomatous nature of the cupping. The recent loss of vision, coming on twenty-two years after the poisoning, could not be related to the original cause. The history of excessive exposure to the noxae of tobacco and to ethyl alcohol, the possible presence of avitaminosis and the exclusion of other probable causes, together with the prompt and spectacular recovery following the use of vasodilators and the vitamin B complex, are adequate reasons for the diagnosis of toxic amblyopia due to ethyl alcohol and tobacco superimposed on an old atrophy of the optic nerve due to methyl alcohol. Whether the atrophic nerve is more susceptible than the normal nerve to toxic influences is an interesting question.

DACRYOCYSTORHINOSTOMY

SOME INTERESTING POINTS ON THE OPERATION

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Two great achievements in the field of ophthalmologic surgery, namely, transplantation of the cornea and plastic dacryocystorhinostomy, were accomplished in the course of the last two decades. Keratoplasty rapidly entered the ophthalmologic practice owing to the extraordinary novelty of the operation, the favorable propaganda of the nonmedical press, the commentaries of the laymen and the eagerness of the blind all over the world who, in their ignorance, asked oculists to perform the operation on them. This was not the case with dacryocystorhinostomy, which has not as yet been accepted by the majority of surgeons of the Western Hemisphere, in spite of the fact that it was devised and technically complete many years before keratoplasty, a fact which tends to bring discredit on ophthalmology.

In the interest of ophthalmologists' professional prestige and their duty of selecting for patients the proper treatment for a given condition and because of all physicians' feeling for humanity, the systematic performance of mutilating, antiphysiologic operations, such as complete removal of the lacrimal sac, may be forbidden. Such an operation condemns a patient to suffer, all through life, with lacrimation and its unpleasant consequences.

HISTORICAL CONSIDERATIONS

The idea of draining the purulent material retained in the lacrimal sac into the nasal fossae dates back to the epochs of Celsus, Galenus and Paulus Aegineta, who empirically perforated the bone with the hot cautery.[1] Treatments more reasonable than that were developed later, when the anatomy and physiology of the lacrimal apparatus were better known. Dominique Anel invented catheterization during lavage of the lacrimal sac at the same time that Woolhouse, an Englishman, in 1713,

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Read at the first Pan-American Congress of Ophthalmology, Cleveland, Oct. 11, 1940.

1. Dusseldorp, M.; Courtis, B.; Damel, C. S.; Gallino, J. A.; Malbrán, J.; Re, B. V., and Moulié, H. B.: Vías lagrimales, Cong. argent. de oftal. (1936) **1**:23, 1937.

performed operations for the free passage of tears through a hole in the bone. The latter did not teach his technic to his colleagues, because of a certain egotism, which was prevalent in that epoch. His pupil Plathner in 1724 followed the same egotistical conduct as the teacher, and the operation passed into oblivion. Two centuries later, in 1904, Toti, a contemporary Italian rhinologist, revived the subject and published the technic of dacryocystorhinostomy, which consists in perforating the lacrimal bone and resecting two portions of the nasal mucosa and of the lacrimal sac in order to form a communication between the lacrimal sac and the middle meatus of the nose. West, in 1911, described a technic of dacryocystorhinostomy by the intranasal route, which later was modified by Polyak and others. The reports of these operations were followed by publication of many articles which were intended to prove the advantage of the exclusive extranasal route over the exclusive intranasal one, or vice versa. The controversy about the Toti and West-Polyak technics was harmful to the development of dacryocystorhinostomy and, although it sounds paradoxic, was responsible for the stagnation of the subject for fifteen years. The reason for the lack of agreement on the technic was that neither of the procedures was logical, and so dacryocystorhinostomy failed.

All the cavities and closed spaces of the body are properly lined with epithelial tissue, and in order to obtain permanent drainage of the lacrimal ducts in the manner in which it was proclaimed by Toti, West and Polyak, spontaneous production of epithelial proliferation to unite the wounds of the nasal mucosa and of the lacrimal sac is necessary. Frequently the operation failed because of obstruction of the perforated lacrimal bone by a newly formed growth.

In order to avoid this complication, Ohm, of Germany, had the idea, in 1912, of suturing the two mucosae, making the operation an entirely plastic one. However, it is interesting to note that Ohm's work made no impression even in Germany, and plastic dacryocystorhinostomy met with indifference.

Dupuy-Dutemps and Bourguet, of Paris, feeling the need of as perfect an anastomosis as possible between the nasal mucosa and the lacrimal sac, performed an operation of this type for the first time on Nov. 28, 1919 and reported it in 1920 and 1921, after which the operation became better known.[2]

In this article the numerous modifications of the technic of dacryocystorhinostomy which have been introduced will not be discussed. They are to be blamed for the present lack of a more liberal use of the operation, since they have been the cause of the perplexities and fear of surgeons

2. Dupuy-Dutemps, L., and Bourguet: Procédé plastique de dacryocystorhinostomie et ses résultats, Ann. d'ocul. **158**:241, 1921.

not acquainted with the operation. It is sad to see that even at present modifications are offered which evidently constitute retrogression, since the most necessary and important feature of the operation, namely, the mucosal suture, is eliminated by these technics.

The conception that dacryocystorhinostomy is an eminently plastic operation must remain unchanged and classically established. Thus, illogical operations will no longer be attempted, and all procedures which do not employ the mucosal sutures will be renounced. All the intranasal technics must be rejected. They are performed blindly, and mucosal sutures cannot be executed. The extranasal operations without anastomosis of the mucosae also must be absolutely rejected. The success of the last-mentioned operations is exclusively due to the spontaneous establishment of an epithelial communication between the two mucosae which is not made by the surgeon. It is both antisurgical and unethical to trust to luck, since the cure of the patient and the good reputation of the surgeon depend on the results of the operation. It is also unjust to provoke diffidence toward an operation the abandonment of which means diminished possibilities of its application to other patients who can be benefited by it. Plastic dacryocystorhinostomy through the external route is the only sound and safe operation. It should be the interest of all surgeons to improve the operation without becoming entangled in the technic of operations of only historical value, such as that of the Toti-Mosher type.

COMMENT

Plastic dacryocystorhinostomy is a permanent accomplishment of medical science. The technic of the operation is complete; the results are extraordinarily satisfactory, and the rate of success, in accordance with all the statistics, is as high as 95 per cent. The operation has precise indications and clearly established contraindications. It is harmless and is performed with local anesthesia. The postoperative course is short and without complications. The technic is not difficult. When the surgeon has performed the operation two or three times he feels more confident than when he performs any of the common operations of ophthalmologic surgery, such as operations for cataract and glaucoma, keratoplasty and some others, all of which may be followed by grave complications which never develop after dacryocystorhinostomy. Why, then, are the surgeons who have performed this operation so few? One of the reasons for the lack of popularity of the operation is that the technic is difficult to learn from mere reading of articles, even if their illustrations are good. It is an operation that, in spite of being studied with a cranium in the hand, seems a priori to be difficult to execute and which, according to Basterra Santacruz, should be learned by observation while others perform it. Another reason is the hesitation and perplexity of surgeons because of the confusion caused by the existence of so many

modifications and different names making the variations in technic appear as different operations. Aside from these reasons, dacryocystorhinostomy is an operation recently put into practice. It was not taught to students of ophthalmology until about fifteen years ago. Before that the students learned from their teachers to remove lacrimal sacs, and as ophthalmologists they continue to do so by mere routine.

The execution of the operation is wearisome and time consuming as well, as it may take twenty minutes in some cases and an hour in others. Many well known surgeons do not attempt to perform the operation under the pretext that the patients do not like it. What really happens is that they do not offer the benefits of the operation to the patients because they are not acquainted with it. The wearisome technic of the operation is not to be considered if the results are satisfactory. It is a duty of oculists, as well as of all surgeons, to meet the most difficult problems of their practice. They selected their profession for the benefit of the patients, and their aim is unfulfilled if, because of ignorance or lack of interest in solving intricate surgical problems, they perform an improper operation from which the patient is condemned to an incurable condition, such as lacrimation from the removal of the lacrimal sac. What would be thought of a general surgeon who would deprive a patient with grave gastric ulcer of the benefits of a gastroenterostomy because of the wearisome technic? By analogy, what has already happened with gastrectomy will happen with dacryocystorhinostomy. The former operation was at first reserved for privileged surgeons, whereas nowadays it is done routinely in all hospitals.

During the months I spent in the United States in 1929 visiting important ophthalmologic clinics, including those of New York, Philadelphia and Baltimore, I did not see a single plastic dacryocystorhinostomy performed. I was surprised both by the lack of interest in dacryocystorhinostomy and by the tranquillity with which well known surgeons removed the lacrimal sac in cases of chronic dacryocystitis without realizing the mutilation from the operation. By that time oculists as well known as Dupuy-Dutemps [3] (of Paris, France), Basterra Santacruz [4] (of Madrid, Spain), Gómez-Márquez [5] (of Barcelona, Spain) and others each had statistics on more than a thousand operations of this type. This happened in our hemisphere as well as in Europe. Dupuy-Dutemps, in 1932, reported to the Society of Ophthal-

3. Dupuy-Dutemps, L.: Statistique de mille dacryostomies plastiques, Bull. Soc. d'opht. de Paris, 1931, p. 392.

4. Basterra Santacruz, J.: Dacriocistorinostomía, An. de oftal. **2**:675, 1926; Arch. de oftal. hispano-am. **27**:110, 1927.

5. Gómez-Márquez, J.: Los efectos lejanos de la dacriocistorinostomía y las bases fundamentales de esta operación, An. de oftal. **3**:657, 1927; Arch. de oftal. hispano-am. **28**:197, 1928.

mology of Paris the results of twelve hundred operations and stated that plastic dacryocystorhinostomy was entirely unknown in England and was performed by only a few surgeons in Paris.

INDICATIONS AND CONTRAINDICATIONS

Plastic dacryocystorhinostomy is the operation of selection in all cases of chronic dacryocystitis with empyema and mucocele which is not cured by simple conservative methods and which from the time of Berlin, in 1866, to the present, surgeons have treated by removal of the lacrimal sac. The latter operation, which is mutilating, should be reserved for very old patients and for patients with tumor or tuberculosis of either the lacrimal sac or the nasal mucosa. Ozena, trachoma, lacrimal fistula and serpiginous ulcer of the cornea do not contraindicate the operation. It should not be performed if the upper lacrimal canaliculi or lacrimal points are occluded. Traumatic and nontraumatic stenosis of the lower lacrimal ducts is a major indication for the operation.

TECHNIC

Instruments to be Used.—The instruments are those commonly used for removing the lacrimal sac, aside from those for osteotomy, namely, hammer, chisel and Kerrison's biting forceps. Some surgeons (Gómez-Márquez [6] and Arruga [7]) prefer a crown trephine, similar to that which is used by dentists. Others (Gutzeit,[8] Basterra Santacruz [9] and Hernández Arias, of Cuba) prefer dental burrs.

Preparation of the Patient.—It is advisable to make a roentgen examination of the lacrimal sac with iodized oil or some other opaque substance. However, the roentgen examination of the sac as well as the report of the rhinologist on the conditions of the nasal cavity may be dispensed with. The tests of coagulability of the blood will be informative concerning hemophilia, and at all events it will be advisable to administer a blood coagulant and calcium chloride before the operation.

Anesthesia.—Local anesthesia is obtained with a 2 per cent solution of procaine hydrochloride and a 1: 1,000 epinephrine solution. I prefer the technic of Gómez-Márquez,[10] by which the nasociliary innervation is

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6. Gómez-Márquez, J.: Algunas consideraciones sobre los diversos tiempos de la dacriocistorinostomía, Arch. de oftal. hispano-am. **31**:147, 1931.

7. Arruga, H.: Surgical Treatment of Lacrimation, Arch. Ophth. **19**:9 (Jan.) 1938.

8. Gutzeit, R.: Ein neues Knocheninstrumentarium zur Toti-Operation, Klin. Monatsbl. f. Augenh. **84**:92, 1930.

9. Basterra Santacruz, J.: Consideraciones sobre la dacriocistorinostomía extranasal, Arch. de oftal. hispano-am. **36**:208, 1936.

10. Gómez-Márquez, J.: Contribución a la dacriocistorinostomía, Arch. de oftal. hispano-am. **25**:473, 1925.

blocked, the tissues are infiltrated and there is a direct effect on the lacrimal sac. The nasal mucous membrane is anesthetized with a 10 per cent cocaine hydrochloride and 10 per cent pontocaine hydrochloride solution with a cotton swab introduced through the nostril into the nasal fossa of the same side. It is very useful to give the patient one or two injections of morphine and atropine a few minutes before beginning the operation.

Incision.—The same incision is made as for the removal of the lacrimal sac. It should be done deeply to the bone and preferably near the inner canthus to avoid meeting the angular artery and also to minimize the scar. A retractor of the mastoid type, which, according to Gómez-Márquez, is far better than others, is then put in place. The soft tissues are rapidly separated by means of either a rasp or Farabeuf's bone scraper up to the lacrimal crest, which is an important guiding point. The palpebral ligament is cut, and under it the lacrimal fossa, containing the lacrimal sac, is identified. The sac is carefully retracted by means of a small piece of gauze which is pushed with the fine curved forceps.

Trephining of Bone.—This procedure is the most wearisome and time-consuming step. Trephining must be performed with the utmost precaution, regardless of whether it is done with the hammer and chisel, the burr or the trephine, because the nasal mucous membrane is immediately behind the bone and the success of the operation depends on the preservation of the membrane. As a rule, the opening is enlarged by means of Citelli's or Kerrison's rongeurs. Osteotomy must be large, larger in its vertical axis but without passing above the lacrimal sac. This is unnecessary and involves the danger of penetrating the ethmoid cells. It is best to trephine below this region in the area of the naso-lacrimal duct, thus avoiding tissue which might fill the opening. One must remember individual anatomic differences. An ethmoid aberrant cell should not be mistaken for the nasal cavity. When trephining is performed, the swab in the nasal fossa should be removed, as it prevents retraction of the membranes.

Mucosal Flaps.—The nasal mucous membrane is first exposed and then observed with the aid of an applicator, which is introduced in the nasal fossa through the nostril. The nasal wall of the lacrimal sac is incised from top to bottom with a bistoury, and the incision is finished with the scissors. In order to make sure of incising the lacrimal sac it is advisable to pass a fine catheter through the inferior lacrimal canaliculus, being careful not to injure it. After examination of the cavity of the sac, the nasal mucous membrane is incised also from top to bottom, after which other perpendicular incisions are made which can permit cutting of two quadrangular flaps. When the latter cannot be cut, the

simple technic of Basterra Santacruz can be resorted to.[11] It consists in cutting only a strong anterior flap of the nasal mucous membrane (the so-called flap in the form of a C with anterior hinge), which is directed outward and forward in order to suture it to the anterior edge of the wound of the sac, after which the posterior portion of the sac is resected. When possible, it is advisable to cut and suture the flaps according to the technic of Dupuy-Dutemps [12] and Bourguet, which is proper and most logical. The sutures are made with no. 000 chromic catgut or fine silk, traction on the mucous membranes being avoided as it may tear the membranes, especially that of the lacrimal sac. Because of the deepness of the region, the sutures are knotted with pincers, the retractors having been previously removed.

Suture of the Skin.—Sutures are made in the common manner, taking care to pass catgut or silk near the edges of the wound in order to obtain good healing and to prevent the formation of operative epicanthus.

Postoperative Course.—The postoperative course is most simple. The dressing is changed every twenty-four hours. Rest in bed or in an arm chair is advised. The patient is instructed not to blow his nose and not to be alarmed if small nasal hemorrhages occur. He should be kept in the hospital for two or three days after the operation. A light lavage with physiologic solution of sodium chloride is made through the lacrimal point three days after the operation and is repeated every two days. The stitches in the skin are removed on the fifth day. Failure of the operation is possible only when the technic is faulty, and it becomes evident within the first week. After the first week the possibilities of failure are exceptionally rare, and by the end of the month the results of the operation can be considered permanent.

Complications.—Surgical complications consist of small hemorrhages, presence of ethmoid cells and wounding of the nasal mucosa necessitating the cutting of irregular flaps. Postoperative complications are rare. The inflammatory reaction generally is mild, although it may be more acute. Abscess formation in the nasal septum was a postoperative complication in a case observed in the Calixto García hospital. Marín Amat [13] reported a case with late grave hemorrhage. Keloid scars, palpebral retraction and eversion of the lacrimal points are infrequent complications.

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11. Basterra Santacruz, J.: Dacriocistorinostomía. Simplificación de mi técnica, Arch. de oftal. hispano-am. **26**:385, 1926.

12. Dupuy-Dutemps, L.: Dacriocistorinostomía plástica, An. de oftal. **1**:253, 1925.

13. Marín Amat, M.: Una complicación grave y tardía de la dacriocistorinostomía, Arch. de oftal. hispano-am. **31**:629, 1931.

SUMMARY AND CONCLUSIONS

The only rational and safe dacryocystorhinostomy is the one in which the union of the mucous membrane of the lacrimal sac and of the nose is effected by the surgeon. The operations without this indispensable feature, namely, all intranasal operations and the large number of technics which have been reported for the purpose of simplifying dacryocystorhinostomy by omitting the mucosal sutures, should be rejected. Plastic dacryocystorhinostomy has a classic and entirely definite place in ophthalmic surgery. Ophthalmic surgeons should be well acquainted with the operation, and it should be as popular in ophthalmic practice as the operations for cataract, glaucoma, strabismus and similar diseases.

The technic is difficult to learn but easy to conquer after it is learned. Once the surgeon performs the operation, its satisfactory results and harmlessness stimulate him to resort to it in other cases, and after performing it a few times he becomes a partisan for the operation.

Plastic dacryocystorhinostomy is indicated in all cases of chronic dacryocystitis in which the conservative methods in common use cause no improvement and in which the lacrimal canaliculi and points are patent. It is the only advisable operation for the surgical treatment of lacrimation due to traumatic or nontraumatic obstruction of the lower lacrimal ducts.

The indications for the removal of the lacrimal sac should be limited to the utmost, which will result in the preservation of the natural prestige of ophthalmology. In the present state of development of ophthalmology, it is unpardonable to condemn a patient, especially a young one, to suffer all through life from lacrimation and its unpleasant consequences, when there is a harmless and rational plastic operation available.

DISCUSSION

Dr. Ralph O. Rychener, Memphis, Tenn.: Dr. Yanes has demonstrated by the motion pictures that this operation can be done skilfully in a clean field and with a minimum of labor. If the test of a good surgeon is his ability to make a difficult procedure look easy, Dr. Yanes has clearly fulfilled all of the requirements.

I am in accord with the theme of this paper, as for more than ten years I have been urging the substitution of some form of dacryocystorhinostomy for the older and more generally practiced operation of removal of the lacrimal sac. Except in the very aged and debilitated patient, the former should always be attempted, for after all there is nothing which can compare with the restoration of normal physiologic function. In the few cases in which the operation results in failure, another attempt may be made, or the sac may always be removed if for some reason it may seem imperative to do so. It is difficult for me to visualize any such necessity.

Many operations and technics have been devised in the desire to reopen the normal drainage canal into the nose, and these may be broadly

grouped under these headings: (1) the external operation, with sutures, popularly called the Dupuy-Dutemps and Bourguet operation; (2) the internal, or Halle-West, operation; (3) the combined, or Toti-Mosher, procedure; (4) the transplantation method, and (5) the giant probings with insertions of a metal style or hollow tube.

As Dr. Yanes has pointed out, all of these except the first are open to legitimate criticism. The combined operation without sutures is often unsuccessful owing to nonunion of the mucous membrane flaps or rapid closure of the bony window. In addition, it requires much unnecessary intranasal surgery. The intranasal operation is entirely outside the ophthalmologist's field and requires the services of a rhinologist, who must be a highly skilled technician. It is not a procedure suitable for the hands of even the average otorhinolaryngologic surgeon. The transplantation method does not allow inspection of the interior of the lacrimal sac, where a pathologic condition may exist to such extent as to nullify the operation. Giant probings have the advantage of speed and facility, but on withdrawal of the style or tube, the opening may again close.

The external dacryocystorhinostomy with sutures is an operation which any one with any surgical training can learn to do. The instruments required are simple and available in the cabinet of any one who does otorhinolaryngologic surgery. The experience of Dr. Yanes with the electrically driven trephine for making the opening between the anterior lacrimal crest has been similar to mine in that the nasal mucous membrane was frequently torn in such a way as to make plastic repair difficult. After a thorough trial with the trephine I have returned to the use of the old-fashioned mallet and chisel for removal of the bone. A few points in technic which I have found helpful are: (*a*) illumination of the operative field by an electric head lamp or a reflecting mirror and (*b*) control of venous and capillary bleeding by the suction tip. The time of operation can be materially shortened by the use of small, half-curved needles swaged onto 000 chromic catgut; I have used these with much satisfaction for the past two years. With a small mosquito forceps these needles may be inserted in the mucous membrane flaps with much more ease and facility than is possible with all of the various and fancy needles and needle holders that have been devised for this purpose. By including the periosteum and subcutaneous tissue over the nasal bone in the anterior sutures, the anterior wall of the newly formed canal is elevated out of the bony window and function begins at once, as the canal is patent. A subcuticular silk or horsehair suture makes a beautiful skin closure with an imperceptible scar.

I have performed this operation successfully in patients with an age range of 3 years to 75, and an elderly woman of the latter age after five years still feels that the relief afforded her by this operation was the greatest boon of her lifetime. In line with the sentiments expressed by Dr. Yanes, I wish to recommend the external operation with sutures to all surgeons who are not satisfied with their own technics and to urge in particular that it be substituted for the out-moded procedure of dacryocystectomy.

BAND-SHAPED OPACITY OF THE CORNEA ASSOCIATED WITH JUVENILE ATROPHIC ARTHRITIS

REPORT OF A CASE

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Band-shaped opacity of the cornea is usually classified on an etiologic basis into three categories: (1) primary, (2) secondary and (3) traumatic. All ophthalmologists are familiar with the band-shaped opacity of the secondary type, which occurs in degenerated and usually shrunken eyes of old persons. However, there is a second subtype, an extremely rare form, which occurs in children, the great majority of whom have rheumatism in any degree up to infantile polyarthritis.[1] With this type the eyes are otherwise sound.

Ohm [2] in 1910 was the first to record an occurrence of this condition in a child who was suffering with arthritis. Uhthoff,[3] Fuchs,[4] Karsch [5] and others later reported similar cases. Uhthoff in his report stated that in 200,000 cases of ocular disease observed by him there was only 1 such case. Zeeman,[6] in a recent article, reviewed the literature, collected 17 cases and added 5 cases of his own. He emphasized the association of band-shaped corneal degeneration and evidences of low grade iridocyclitis with chronic arthritis in children and referred to the

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1. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 2010.

2. Ohm, J.: Bandförmige Hornhauttrübung bei einem neunjährigen Mädchen und ihre Behandlung mit subkonjunktivalen Jodkaliumeinspritzungen, Klin. Monatsbl. f. Augenh. **48** (pt. 2):243, 1910.

3. Uhthoff, W.: Ein Fall von typischer bandförmiger Trübung der Hornhaut auf beiden Augen bei einem achtjährigen Mädchen mit teilweiser erhaltener Sehkraft und hinteren Synechien, Klin. Monatsbl. f. Augenh. **60**:11, 1918.

4. Fuchs, E.: Ueber gürtelförmige Hornhauttrübung, Klin. Monatsbl. f. Augenh. **61**:10, 1918.

5. Karsch, J.: Ueber sekundäre bandförmige Hornhauttrübung bei der Polyarthritis chronica leukocytotica (Still) und lymphocytotica (Rhonheimer), Arch. f. Augenh. **110**:106, 1936.

6. Zeeman, W. P. C.: Band-Shaped Degeneration of Cornea and Chronic Arthritis in Children, Nederl. tijdschr. v. geneesk. **84**:134, 1940.

combination as a definite syndrome. He stressed the need of careful ocular examination of children suffering with chronic rheumatism. It is noted that the American literature has neglected this condition, and because its occurrence is sufficiently rare the following case, which was observed recently in the Cleveland City Hospital, is submitted.

REPORT OF CASE

A. W., a white boy 13 years of age, was admitted to the Cleveland City Hospital on Nov. 20, 1940 for treatment of an atrophic arthritis involving the joints of his extremities, recurrent attacks of fever and defective vision of both eyes. The onset of his articular symptoms occurred at the age of 4 years, when his left knee first became painful, stiff and swollen. The condition gradually involved the other joints of the extremities. About six months after the onset of the initial articular symptoms his sister noticed some "white spots" in the cornea of his left eye. A competent ophthalmologist found that there were many small grayish white opacities in the cornea, in the exposed portion of the palpebral fissure. Low grade iridocyclitis was present. The right eye was normal. Treatment consisted of dilation of the pupil of the left eye. Two years before the present illness, during a routine physical examination, the right eye was found to be similarly involved.

The only ocular symptom experienced by the boy was blurring of vision. Gross inflammation of the eyes was never observed. Until about one year before he came to the hospital he had been able to read ordinary print and work on his stamp collection without any help.

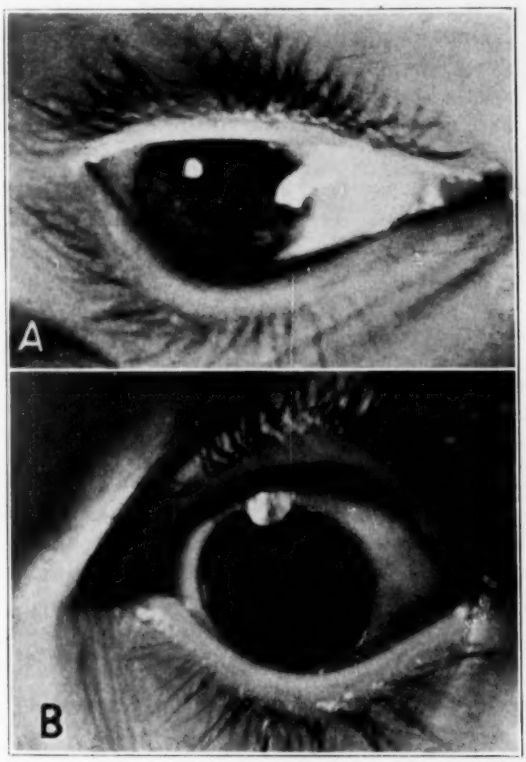
Physical Examination.—The physical appearance of the boy was striking. He was obese and had a somewhat feminine appearance. There were complete ankylosis and fusiform enlargement of the knee and ankle joints and partial ankylosis and enlargement of the joints of the wrists and fingers. Some stiffness of the neck was present. The cervical and axillary lymph nodes and the spleen were enlarged.

Laboratory Examination.—Roentgen examination of the joints showed characteristic osteoporosis of the bones near the articular surfaces of nearly all the joints of the extremities. The joints were enlarged. There was no spurring of the articular surfaces. Films of the chest did not show any evidence of tuberculosis.

An intravenous congo red test with 20 cc. of a 1.5 per cent solution of the dye gave a strongly positive reaction, indicating the presence of generalized amyloidosis. Study of the blood picture and chemical analysis of the blood did not show any significant changes. The Wassermann reaction was negative.

Ocular Examination.—Vision in the right eye was reduced to counting fingers at 1 meter, and that of the left eye was limited to recognition of hand movement at 0.5 meter. The lids and conjunctivas were normal. The eyeballs were of normal size. In the palpebral interspaces of each cornea was a typical sharply demarcated gray band-shaped opacity 3 mm. broad, extending laterally just short of the limbus and vertically to just above the middle of the pupil, such as is frequently seen in blinded or atrophic eyes. The opacity was slightly more dense in the central area than in the periphery. The sensibility of the cornea was decreased in the region of the band-shaped opacity but was well maintained in the portion above and below it. The fundi could not be visualized because of the corneal opacities and the partially occluded irregular pupils. Tension in the right eye was 22 mm. of mercury (Schiötz), and in the left, 19 mm.

Biomicroscopic examination of the right eye showed that the epithelial surface was slightly roughened. The translucent band was composed of numerous minute gray dots with about ten or twelve large white opacities scattered irregularly in it. There were several clear spaces between the opacities. The opacities were situated just beneath the epithelial layer and extended through Bowman's membrane. There was no evidence of inflammation in the deep layers of the cornea, and there were no deposits on the posterior surface of the cornea. The anterior chamber was of normal depth. The iris showed little evidence of atrophy, and the pupillary margin was bound to the anterior lens capsule in several areas by threadlike posterior



A, photograph of the right eye, showing the band-shaped opacity. *B*, photograph of the left eye.

synechiae. The pupillary space was partially occluded by deposits of brownish pigment granules. The left eye was the same as the right except that the changes were slightly more advanced and there were more large white opacities.

Use of atropine sulfate solution and later of atropine powder and epinephrine hydrochloride 1 : 100 was ineffective in dilating the pupils. Removal of the opacities by scraping the corneal and optical iridectomy was contemplated, but permission for operation was refused by the patient's family.

Two hours after the intravenous injection of the congo red solution biomicroscopic examination of the cornea showed that some of the minute gray dots

nearest the limbus of the right eye stained a faintly visible pink. Instillation of congo red and later of iodine solution into the cul-de-sac did not cause any additional staining of the opacities.

COMMENT

Little is known of the pathogenesis of band-shaped opacity of the cornea, although the pathologic changes have been fully studied. A disturbance in nutrition with resultant diminution of the ability of the cornea to withstand external injurious influences is considered an essential factor. The changes associated with the various types of band-shaped opacity are essentially the same except in degree, and with the primary type and the type just described the eye is otherwise healthy. In the early stages there is deposition of amyloid or hyaline degeneration. Later there are deposits of phosphates and carbonates of lime and of small quantities of sulfur and silicon.[7] The staining of some of the opacities near the limbus of the right eye after the introduction of congo red into the vascular system offered presumptive evidence of the presence of either amyloid or hyaline material in the cornea. However, since operation was refused and the opacities could not be removed for direct examination, the exact nature of this material could not be determined. The fact that amyloid deposits in the viscera and lymph nodes are a common feature of juvenile chronic arthritis makes the assumption of some authors that the deposition of amyloid in the cornea is associated with deposits of amyloid elsewhere seem quite convincing.

SUMMARY

A case of band-shaped opacity of the cornea associated with chronic arthritis in a 13 year old boy is reported. The combination of this degenerative change of the cornea with chronic arthritis in children is regarded by some authors as a definite syndrome.

The type of corneal dystrophy described in this case occurs in comparatively healthy eyes.

7. Sisson, R. J.: Band Keratitis, Am. J. Ophth. **17**:222, 1934.

RETINAL HEMORRHAGES IN THE NEWBORN

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Systematic studies of the fundi of large numbers of newborn infants have left no doubt as to the frequency of hemorrhages from the retinal vessels. In an effort to establish a correlation between this condition and the circumstances of labor, the accumulated statistics on the subject have been reviewed and a series of 498 newborn infants have been examined and studied with reference to labor.

According to Ehrenfest,[1] the first recorded observations of the fundi of the newborn were those of Jaeger [2] in 1861. In an elaborate paper published in 1909, Stumpf and von Sicherer [3] presented a history of the investigation of the subject to that date and reported the results of their own study of a large number of cases. Their most important findings were as follows: 1. The corpus vitreum was always normal. 2. The eye-ground of the newborn child in general resembled that of the adult, although as a rule all the colors were lighter. 3. If the examination was made soon after birth, a distinct venous stasis was common. 4. The papilla usually appeared normal, but, as was emphasized also by Jaeger and Koenigstein, it was dark gray or bluish gray in some cases. 5. The usually coexisting reddish discoloration at the scleral edge with fine, red, radiating hemorrhagic lines indicated that the darkening of the papilla was the result of a hemorrhage in the subvaginal space of the dural sheath; within a few days the blood was resorbed and the color normal.

FREQUENCY OF RETINAL HEMORRHAGES

Retinal hemorrhages in various portions of the eye have constituted the most striking ophthalmoscopic finding in the newborn. Reports of their frequency vary widely, however, from the figures of Koenig-

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1. Ehrenfest, H.: Birth Injuries of the Child, New York, D. Appleton and Company, 1922, pp. 34 and 66.

2. Jaeger, E.: Ueber die Einstellung des dioptrischen Apparatus im menschlichen Auge, Vienna, L. W. Seidel u. Sohn, 1861.

3. Stumpf, M., and von Sicherer: Beitr. z. Geburtsh. u. Gynäk. **13**:408, 1909.

stein,[4] who found them in only 10 per cent of cases, to those of Coburn,[5] who found them in 46 per cent of cases. This variation is probably due to differences in the length of time that the various observers allowed to elapse between the birth of the infant and the examination of the fundi. The earlier the examination is made, the more hemorrhages are likely to be seen, since small hemorrhages readily absorb and quickly disappear. Von Sicherer's figures (35 per cent of 65 cases) are based on observations made within twenty-four hours of birth.

CAUSAL FACTORS

Retinal hemorrhages in the newborn probably represent the result of combined abnormal fragility of the walls of the vessels and increased intravascular pressure. Thompson and Buchanan [6] and Wolff [7] expressed the belief that they are the result of increased blood pressure due to obstruction of the fetal circulation, and Bahn,[8] that they might be attributed to diapedesis from the veins, either in the course of a slight asphyxiation or because of the interval between the cessation of the maternal circulation and the full action of the child's heart. Paul [9] and other investigators stated that loops of cord about the neck cause retinal hemorrhages, but Stumpf found them in only 14 per cent of 63 babies born with this condition. Wolff and Stumpf called attention to asphyxia as a cause of retinal hemorrhages; Stumpf reported them in 6 of 18 cases, or 33 per cent.

Contracted pelves have been considered a contributing factor in retinal bleeding by several authors, but Stumpf stated that the condition occurs with the same frequency in infants delivered normally. Eades [10] expressed the same opinion and reported that only 20 per cent of his newborn infants with retinal hemorrhages were delivered through contracted pelves; Jacobs [11] found this to be the case in 10 per cent of his cases, Seissiger [12] in 33 per cent, and both Hoffman [13] and Paul in 50 per cent.

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4. Koenigstein, L.: Med. Jahrb., 1881, p. 47.
5. Coburn, E.: Arch. Ophth. **33**:256, 1904.
6. Thompson, W. E., and Buchanan, L.: Tr. Ophth. Soc. U. Kingdom **23**: 296, 1903.
7. Wolff, B.: Ophthalmoscope **5**:484, 1907.
8. Bahn, C. A., in discussion on Signorelli, J.: New Orleans M. & S. J. **79**: 454, 1926.
9. Paul, cited by Ehrenfest.[1]
10. Eades, M. D.: New England J. Med. **201**:151, 1929.
11. Jacobs, M. W.: Retinal Hemorrhage in the New-Born, J. A. M. A. **83**: 1641 (Nov. 22) 1924.
12. Seissiger, J.: Arch. f. Augenh. **100-101**:302, 1929.
13. Hoffman, A. A.: J. Iowa M. Soc. **16**:25, 1926.

Stumpf and von Sicherer stated that syphilis plays a part in producing retinal hemorrhages, but other observers have found as many hemorrhages in nonsyphilitic as in syphilitic infants, and Jacobs and Eades found none in any of their syphilitic infants.

Ehrenfest stated that severe intracranial pressure at the time of birth affects the eye through hypertension in the supravaginal space about the nerve and through impeded venous return of the retinal circulation, which drains into the cavernous sinus. According to Ehrenfest, Schleich's [13a] theory that the congestion is the effect of compression of the cavernous sinus was substantiated by von Sicherer's finding that in the more common left anterior occipital presentation the hemorrhage is more likely to occur in the right eye, or, if bilateral, is more pronounced in the right eye, and that just the opposite situation obtains if there is right anterior presentation. This would seem to indicate that the ocular lesion would be found more frequently on the side on which the sinus is exposed to greater pressure. Juler,[14] however, did not find retinal hemorrhage more commonly in the right eye following left anterior occipital presentation or the reverse following right anterior occipital presentation.

According to Whitnall,[15] the central vein of the retina usually opens separately into the cavernous sinus but sometimes joins the superior or even the inferior ophthalmic vein instead and has always at least one side connection, commonly with the superior ophthalmic vein. This vein is formed in the adult by the continuation of the angular vein at the inner angle of the lids and ends in the cavernous sinus. The inferior vein from the floor of the orbit usually enters the superior vein or the sinus directly and sometimes has communication with the pterygoid plexus through the inferior orbital fissure. Thus, although there may be anastomosis between the central and the angular vein, the central vein usually drains mainly into the cavernous sinus, which is likely to suffer most in the cerebral compression during birth. Juler expressed the belief, therefore, that, regardless of other factors, the production of retinal hemorrhages depends on the venous congestion in the central retinal vein, which is induced by the great intracranial pressure during birth. He concluded that abnormalities of labor which tend to increase the cranial pressure exercise little effect on the incidence of retinal hemorrhages. In this view he was supported by Edgerton,[16] who stated that the main factor

13a. Schleich, cited by Edgerton.[16]

14. Juler, F.: Tr. Ophth. Soc. U. Kingdom **46**:42, 1926.

15. Whitnall, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision, ed. 2, New York, Oxford University Press, 1932, p. 314.

16. Edgerton, A. E.: Ocular Observations and Studies of New-Born, with Review of Literature, Arch. Ophth. **11**:838 (May) 1934.

involved is probably congestion of retinal veins due to increased intracranial pressure arising from the circumstances of labor and that abnormalities of labor do not increase the hemorrhagic tendency unless a high forceps delivery is used.

Naumoff [17] concluded that while the head is being molded during labor the spinal fluid is forced into the subvaginal space of the optic nerve, causing strangulation of the central vein of the retina. Otherwise, Naumoff argued, choroidal hemorrhages would occur as often as retinal hemorrhages, and choroidal hemorrhages at birth are rare. Naumoff himself, however, found retinal hemorrhages in a few cases in which the subvaginal space was entirely absent.

Schleich and von Sicherer concluded that hemorrhages from the small retinal vessels are the result of congestion and are etiologically the same as cerebral hemorrhages.

A STUDY OF FOUR HUNDRED AND NINETY-EIGHT CASES

The fundi of 498 newborn infants have been examined and studied during the past year. The majority of these were observed within forty-eight hours of birth. The best time for making the examination was found to be about thirty minutes before feeding time, when the baby is awakened for a sponge bath and change of clothing and for supplemental feeding if required. A small amount of sucrose or lactose was offered when no supplemental feeding had been prescribed. Five-tenths per cent homatropine hydrobromide was instilled in all cases. Aseptic precautions were taken, and with the assistance of a nurse the examinations were made quickly and satisfactorily.

TYPES OF RETINAL HEMORRHAGES

Retinal hemorrhages may be classified according to three principal types as follows:

1. *Most Common.*—Small, flame-shaped hemorrhages which appear to be superficial to the vessels above and below the disk. In this series they seemed to follow the direction of the arterioles and were confined to the posterior pole of the eye (fig. 1 *A*). Subsequent examinations showed that they were absorbed in from two to five days (fig. 1 *B*). In all probability these bear no relation to subsequent impairment of vision.

2. *Less Common.*—Thin, sheetlike hemorrhages in the nerve fiber layer, spreading out from the optic disk toward the equator. These are less pigmented and may escape the notice of the observer (fig. 7 *A*). In this series they were found to be absorbed in from three to five days. They are believed to cause no impairment of vision.

17. Naumoff, M. P.: Arch. f. Ophth. **36**:180, 1890.

3. *Least Common.*—Dull red, dense, diffusely round hemorrhages (fig. 2 *A*). These are well circumscribed collections of blood showing tiny reflexes at their summits from the light of the ophthalmoscope and are found in or about the macular region. They are about two-thirds

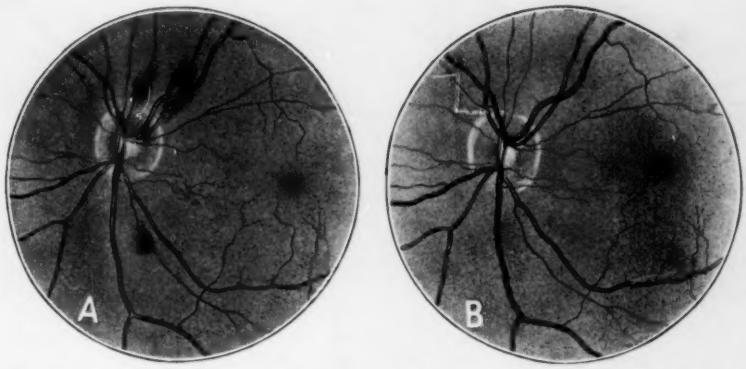

Fig. 1.—*A*, left eye of baby E. D., second child (boy) of a 35 year old mother. Spontaneous delivery took place in nine hours from a right anterior occipital position. There was asphyxia, and artificial respiration was required. Large and small flame-shaped hemorrhages were observed forty-eight hours after birth. There was venous congestion at the disk margin. *B*, left eye on the fifth day after delivery.

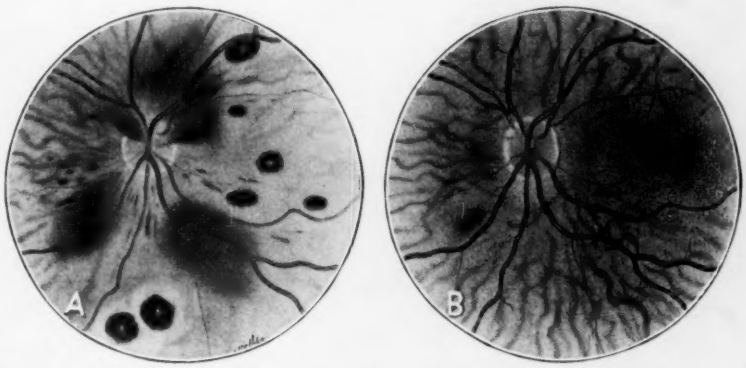

Fig. 2.—*A*, left eye of baby A. T., third child (boy) of a 35 year old mother. Spontaneous delivery took place in twenty hours from a left anterior occipital position. The cord was around the neck once. Eighteen hours after birth all three types of hemorrhages were present. *B*, only slight evidence of hemorrhage in the lower nasal quadrant on the fourteenth day after delivery.

the size of the disk, and are usually not absorbed until from ten to fifteen days after birth (fig. 2 *B*). Juler stated that he found these hemorrhages most commonly in the macular region and that they seemed to be in a

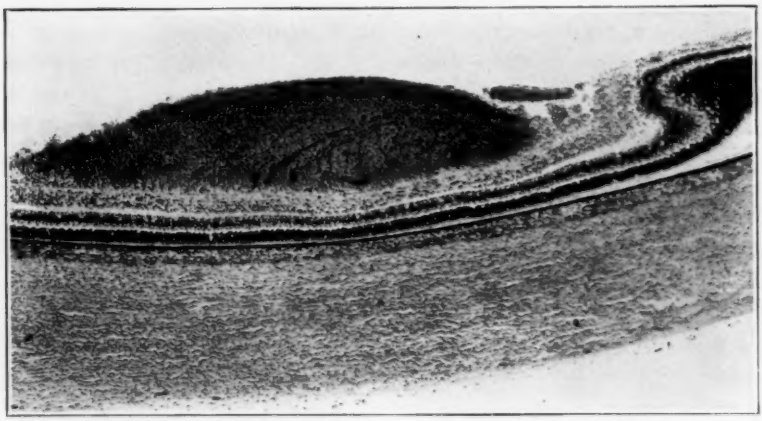

Fig. 3.—Right eye of baby L. O'N., a three week premature boy who lived one and one-half hours. A dark red, well circumscribed hemorrhage is seen in the retina temporal to the macula. The anterior limiting membrane of the retina separates the hemorrhage from the preretinal or subhyaloid space.

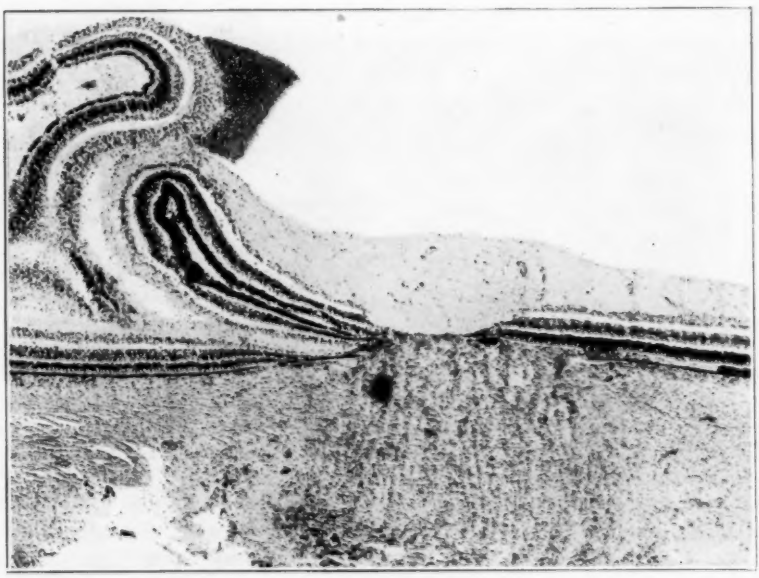

Fig. 4.—Right eye of baby L. O'N. Retinal folds at or near the disk margins are not uncommon and may simulate edema ophthalmoscopically.

plane anterior to the retina; he called them subhyaloid hemorrhages. Richman,[18] on the other hand, described them as definitely preretinal. Microscopic examination in several cases in this series indicated that

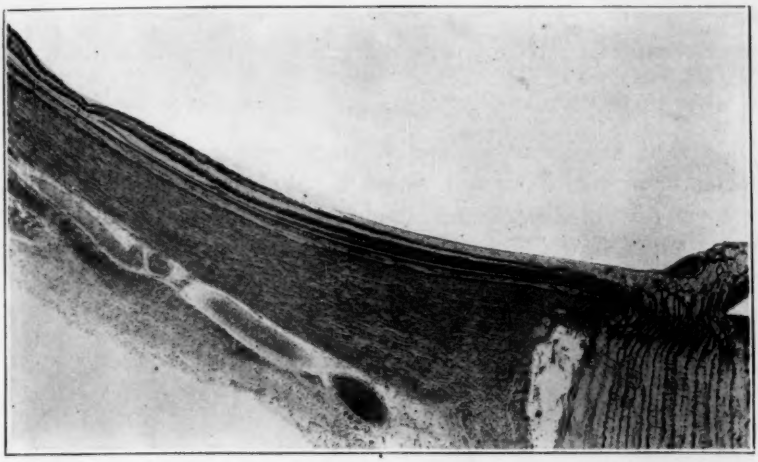

Fig. 5.—Right eye of baby E. D. Enucleation was done on the third day after delivery. There are one hemorrhage between the disk and the macula and three others temporal to the macula. All are well within the anterior limiting membrane and seem to be in the nuclear layer.

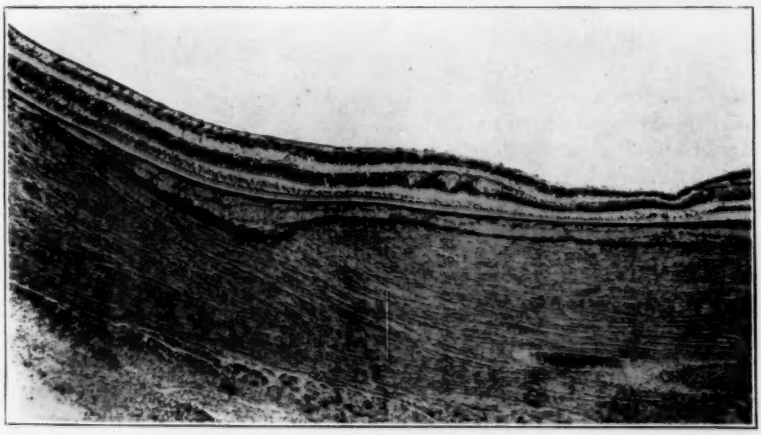

Fig. 6.—Higher magnification of figure 5, showing a diffuse hemorrhage in the nuclear layer temporal to the three small hemorrhages near the macula.

they were in retinal tissue and were separated from subhyaloid and preretinal spaces by the anterior limiting membrane of the retina (figs.

18. Richman, F.: Tr. Internat. Cong. Ophth., 1937 (commun. libres) **4**:76, 1938.

3 and 5). The persistence of this type of hemorrhage in the macular area may produce structural changes which will interfere with vision in later life. Several observers have considered them a possible cause of amblyopia, of amblyopia in a squinting eye, of some cases of congenital nystagmus, and of degenerative changes in the macula. Jacobs, however, expressed the opinion that they do no permanent damage to the eye.

OTHER FINDINGS

Edema of the disk margin and commotio retinae were not infrequent, particularly in eyes examined within a few hours of delivery. These symptoms usually disappeared within forty-eight hours. Microscopic examination of several eyes obtained at birth or soon after revealed the presence of folds in the retina at the disk margins (fig. 4). These may

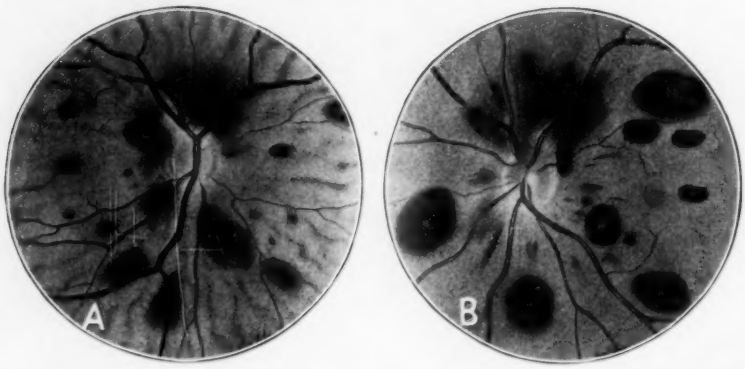

Fig. 7.—A, right eye of baby M. L., the third child (a boy) of a 32 year old mother. Spontaneous delivery occurred in sixteen hours from a right anterior occipital position. The fundus showed large sheets of hemorrhages twenty-four hours after birth. A dark red, well circumscribed hemorrhage was between the disk and the macula. *B*, left eye of baby J. L., the second child (a boy) of a 33 year old mother. Spontaneous delivery occurred in thirteen and one-half hours from a left anterior occipital position. The cord was around the neck once and was cut before delivery of the shoulders. Twenty-six hours after birth the fundus showed many dark red, well circumscribed hemorrhages, a sheet of hemorrhage above the disk and some flame-shaped hemorrhages between the disk and the macula.

have accounted for the edematous appearance of the disk margins in some cases and may or may not have been associated with hemorrhages.

INCIDENCE

In the entire series of 498 cases retinal hemorrhages were found in one or both eyes of 210 babies, or 42.1 per cent (fig. 8). They occurred twice as often among the primiparas (126 of 244 cases, or 51.6 per cent) as among the multiparas (64 of 254 cases, or 25 per

cent), and slightly oftener among the 234 boys (112 times, or 47.9 per cent) than among the 264 girls (98 times, or 37.1 per cent).

Correlation With Delivery Complications (fig. 9).—The incidence of retinal hemorrhages was high in cases in which the delivery was other than normal and spontaneous, with the exception of the 10 cases of cesarean section, in none of which were retinal changes observed. A forceps was used in 148 cases (low forceps delivery more often than high), and in 86, or 58.1 per cent, of these there were hemorrhages. There were 14 breech deliveries, and hemorrhages were associated with

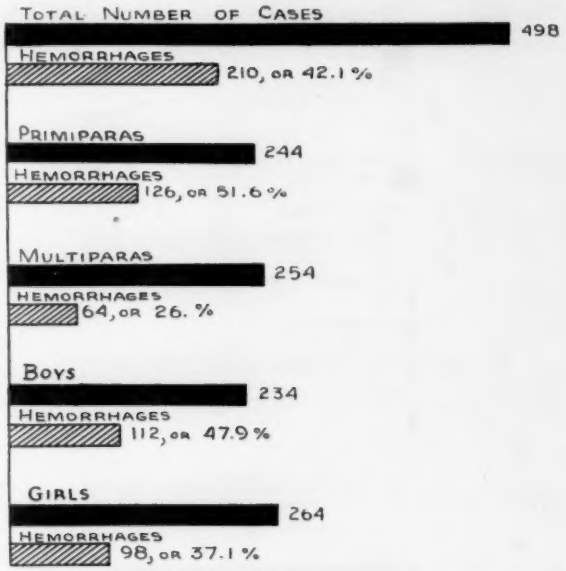

Fig. 8.—Incidence of retinal hemorrhages in the total series of cases.

8, or 57.1 per cent, of them. Asphyxia obtained in 48 cases, in only 12, or 25 per cent, of which there were hemorrhages.

There has been considerable variation in the percentages of hemorrhages reported for babies born with the cord about the neck. According to Jacobs, "Paul blamed strong compression of the jugular vein by a tight coiling of the cord around the neck. But he was answered by Stumpf, who found retinal hemorrhages in only 14 per cent of cases in which the cord was around the neck, but in 24 per cent without the cord anomaly." In this series the cord was around the neck four times in 1 case, three times in 1, twice in 3 and once in 19 (fig. 7 *B*). Retinal hemorrhages were found in 22 cases, or 91.6 per cent.

Correlation With Position of the Head (fig. 10).—In this series the position of the infant's head on entering the birth canal appeared to

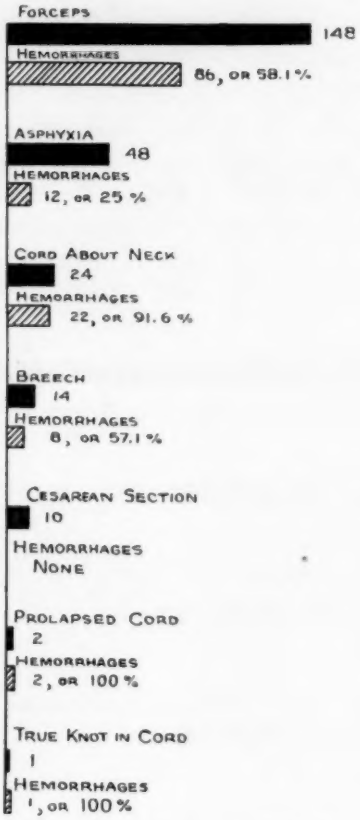

Fig. 9.—Incidence of retinal hemorrhages according to complications of delivery.

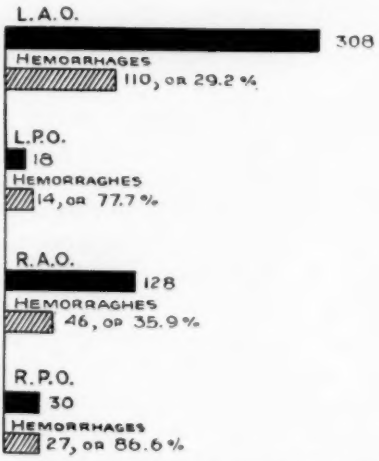

Fig. 10.—Incidence of retinal hemorrhages according to occipital presentation: *L.A.O.*, left anterior occipital; *L.P.O.*, left posterior occipital; *R.A.O.*, right anterior occipital; *R.P.O.*, right posterior occipital.

influence the incidence of retinal hemorrhages. Of the four positions, the left anterior occipital is the most common. This was observed in 308 cases, and hemorrhages were found in 110, or 29.2 per cent. There were 18 cases of left posterior occipital presentation, and 14, or 77.7 per cent, showed hemorrhages. One hundred and twenty-eight babies had a right anterior occipital delivery, and 46, or 35.9 per cent, had hemorrhages. In the 30 cases in which the infants were delivered from a right posterior occipital position, forceps were used in a high percentage, and the labors were long; hemorrhages were found in 26, or 86.6 per cent.

Correlation With Age of Mother (fig. 11).—The incidence of hemorrhages did not vary greatly with the age of the mother. Twenty-

Fig. 11.—Incidence of retinal hemorrhages according to age of the mother.

two babies were born to mothers from 17 to 20 years of age, and 10, or 45.4 per cent, showed hemorrhages. One hundred and twenty-six babies were born to mothers between the ages of 21 and 25, and there were 62, or 49.2 per cent, with hemorrhages. One hundred and ninety-two babies were born to mothers between the ages of 26 and 30, and there were 70, or 37.5 per cent, with hemorrhages. There were 158 babies born to mothers between the ages of 31 and 40, and 62, or 39.2 per cent, showed hemorrhages.

Correlation With Duration of Labor (fig. 12).—The duration of labor, on the other hand, showed a most convincing correlation with the incidence of retinal hemorrhages. One hundred and thirty-four babies were delivered in less than six hours of labor, and 38, or 28.3 per cent, had retinal hemorrhages. Two hundred and two were born after from six to twelve hours of labor, and 74, or 36.6 per cent, of

these showed hemorrhages. Seventy-six were born after from twelve to eighteen hours of labor, and 38, or 50 per cent, showed hemorrhages. Forty-six were born after eighteen to twenty-four hours of labor, and 22, or 50.5 per cent, showed hemorrhages. Twenty babies were born after from twenty-four to thirty hours of labor, and 18, or 90 per cent, showed hemorrhages. Finally, of 22 babies born after more than thirty hours of labor, 22, or 100 per cent, showed hemorrhages. Of these last, 1 was born after a fifty-four hour labor.

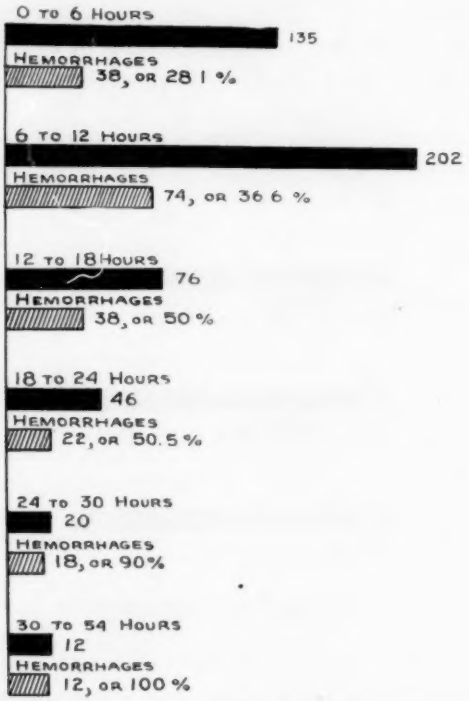

Fig. 12.—Incidence of retinal hemorrhages according to duration of labor.

UNUSUAL CONDITIONS

Two unusual conditions of labor which occurred in this series were a true knot in the umbilical cord in 1 case and a prolapsed cord in another. In both cases there were extensive retinal hemorrhages. In the cord with the true knot the placental pulse could be palpated but was very feeble. The prolapsed cord was clamped and cut before delivery of the head.

Hemorrhages into the vitreous were noted in 3 cases. In 1 the hemorrhage was so extensive that the details of the fundus were indis-

tinct. The child was born with a high Barton forceps delivery from a right posterior occipital position after forty-six hours of labor.

SUMMARY AND CONCLUSIONS

The literature on retinal hemorrhages of the newborn is reviewed. Reports of their frequency vary from 10 per cent to 46 per cent. There seems to be no general agreement as to the exact causes of these hemorrhages, but the main factor is believed to be congestion of the retinal veins resulting from increased intracranial pressure brought about by labor.

In a study of the fundi of 498 newborn infants, retinal hemorrhages were found in 42.1 per cent during the first forty-eight hours after birth.

Most common were small, flame-shaped hemorrhages, superficial to the vessels; less common were thin, sheetlike hemorrhages in the nerve fiber layer, and least common were dull red, dense, diffusely round hemorrhages.

The flame-shaped and the sheetlike hemorrhages are believed to cause no subsequent impairment of vision, but the location of the dense round hemorrhages in or near the macula and their persistence may make them a cause of permanent visual damage.

Edema of the disk and commotio retinae were found frequently and usually disappeared within the first forty-eight hours.

There was positive correlation between the incidence of retinal hemorrhages and of such abnormalities of birth as forceps delivery, breech presentation, protracted labor, cord strangulation and unfavorable position of the head on entering the birth canal. All babies born by cesarean section, however, were free from hemorrhages.

Extensive hemorrhages occurred in 2 infants born with unusual complications: 1 with a true knot in the umbilical cord and 1 with a prolapsed cord. Hemorrhages into the vitreous were noted in 3 cases and were so extensive in 1 that fundus details were obscured.

The 498 infants included in this study were examined by permission of Dr. B. P. Watson, director of the department of obstetrics of the Presbyterian Hospital, and Dr. H. R. Charlton, attending physician in charge of the obstetrics department of the Lawrence Hospital.

CLINICAL OBSERVATIONS ON THE TREATMENT OF SQUINT

J. B. FELDMAN, M.D.
PHILADELPHIA

In 1934 and 1935 I published several articles [1] on the construction of the squint clinic at St. Christopher's Hospital for Children and the technic followed there. Since that time this department has continued to function, independent of the eye dispensary.

This report is based on a study of 56 privately referred patients and 72 dispensary patients. Each group received orthoptic treatment by a trained technician under my supervision. Among the 56 patients referred to my office for private treatment there were only 6 with unilateral squint; the remainder had the alternating type. Four of the latter had alternating divergent squint. The dispensary group consisted of 36 patients with alternating squint, 3 with alternating divergent squint and 34 with unilateral squint.

The larger percentage of patients with alternating squint in both groups is accounted for in part by the fact that, since alternating squint is very difficult to cure, many patients remained over from the previous year. To these were added a number of newly referred patients. Some of the patients with alternating squint also came for postoperative treatment, while others, for one reason or another, preferred prolonged treatment to surgical intervention. In this group of private patients and patients of the clinic, the sexes were equally represented. In 71 per cent the left eye was most often seen to squint.

A previous history of squint in the immediate family was noted in 23 per cent of cases. This was less than the incidence noted by Fink and Bryngelson,[2] who did not limit the history to the immediate family. My records also show only 3 sets of twins in the entire group, as against 10 to 15 per cent of twins observed by Cords (cited by

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From the Squint Clinic of St. Christopher's Hospital for Children.

Read before the Section of Ophthalmology of the College of Physicians of Philadelphia, Feb. 20, 1941.

1. Feldman, J. B.: (*a*) Eye, Ear, Nose & Throat Monthly **12**:501, 1934; (*b*) Orthoptic Treatment of Concomitant Squint, Arch. Ophth. **13**:419 (March) 1935.

2. Fink, W. H., and Bryngelson, B.: Relation of Strabismus to Right or Left Sidedness, Arch. Ophth. **14**:947 (Dec.) 1935.

Travers [3]). There were 2 stutterers, 3 left-handed children and 3 rachitic children.

In 41 per cent of my group squint was noticed before the age of 3 years. The alleged causes of the squint as obtained from the histories included falls, pertussis, convulsions, pneumonia, mastoidectomy and postfebrile conditions.

CONSIDERATIONS PRELIMINARY TO ACTIVE ORTHOPTIC TREATMENT

Refraction should be performed. A number of examinations of each patient is necessary to determine the angle of squint and the correct diagnosis, as well as the major obstacles to be expected in the particular case. These repeated examinations also serve to gain the confidence of the patient and of the parents, so that more cooperation is obtained. The benefits of a correct diagnosis are too well known to require repetition.

Determination of the Angle of Squint.—It was found that the simplest devices by which the angle of squint could be measured in children were the deviometer,[4] the perimeter and the synoptophore. A prolonged test for determining the angle of squint in a young child does not merit the time consumed. A simple method of taking these measurements makes it possible to repeat the examination several times and on different days to assure consistency. For a qualitative estimation, the Hirschberg rule of the relative position of the light reflex on the cornea gives a fair idea of the angle of squint.

Relation of the Angle of Squint to Refractive Error: I was able to confirm the finding of Corrado [5] that a high refractive error is not consistently seen in association with a high degree of squint or vice versa. Thus, in my series it was noted that each of 10 patients with a high refractive error had a squint of less than 15 degrees, while each of 12 patients with squint of over 40 degrees had a low refractive error.

Relation of Vitamin A to Squint.—Shaad [6] cited D'or as noting a relation between lack of vitamin A and squint when a hereditary relation exists. I have not found this association in 35 patients with squint when a hereditary relation did exist. The light threshold was normal as tested by the adaptometer.[7]

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3. Cords, R., cited by Travers, T. A.: The Comparison Between the Visual Results Obtained by Various Methods Employed for the Treatment of Concomitant Strabismus, London, George Pulman & Sons, Ltd., 1936.

4. Worth, C.: Squint, ed. 6, Philadelphia, P. Blakiston's Son & Co., 1929.

5. Corrado, M.: Ann. di ottal. e clin. ocul. **62**:817, 1934.

6. Shaad, D. J.: Binocular Vision and Orthoptic Procedure, Arch. Ophth. **20**:477 (Sept.) 1938.

7. Feldman, J. B.: Instrument for Qualitative Study of Dark Adaptation, Arch. Ophth. **18**:821 (Nov.) 1937.

Visual Fields.—Determination of the visual fields of a young child is difficult and is often thought to give unreliable results. However, it was possible by selecting the more intelligent children to corroborate one of the findings of Braun,[8] in that a group of 7 children with alternating squints had normal fields.

AIDS TO ORTHOPTIC TRAINING

The following useful hints should be borne in mind in examining a patient with squint; they may greatly affect the diagnosis or the course of treatment in the individual case.

1. It should be remembered that the gamma angle may differ in either eye. A large gamma angle ("false squint") should not be treated, surgically or otherwise.

2. The angle of a paralyzed muscle is not fixed but may vary after some time.

3. In an infant, even if the squint appears to be concomitant the condition may be paralytic. Chavasse[9] reported the case of a child 2½ years old with a paralytic external rectus muscle. After six weeks the squint became concomitant and remained so for twenty-two months. Later the muscle again became paralytic.

4. The earlier a squint develops and the longer treatment is delayed, the less is the possibility of cure or even of improvement.

5. The prognosis for a squint of high degree with amblyopia is poor.

6. Great impediments to orthoptic training are the vertical anomalies anisometropia and aniseikonia. For the latter Berens[10] has successfully prescribed iseikonic glasses in various instances.

7. Peter suggested that essential alternating squint is incurable and that the condition may be aggravated by orthoptic treatment.[10]

8. That atropine is useful in the differential diagnosis of accommodative and pseudoparalytic squint was indicated by Guibor.[11]

9. In the etiologic classification of Bielschowsky[12] it was stated that a constant deviation in a squint is an indication that the cause is anatomic. Unsteadiness in the angle of squint is possibly due to excessive nervous innervation.

10. There is no difference neurologically between a unilateral and an alternating squint. An alternating squint may be associated with unilateral fixation, and vice versa.

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8. Braun, G.: Klin. Monatsbl. f. Augenh. **92**:600, 1934.

9. Chavasse, B.: Tr. Ophth. Soc. U. Kingdom **50**:168, 1930.

10. Berens, C., in discussion on Peter, L.: Tr. Am. Ophth. Soc. **33**:26, 1935.

11. Guibor, G. P.: Classification of Concomitant Strabismus: Results Secured in Various Types, Arch. Ophth. **19**:947 (June) 1938.

12. Bielschowsky, A.: The Etiology of Strabismus, in Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1940.

REFRACTION

It is generally conceded that correct refraction is the first requisite in the correct treatment of squint. However, all ophthalmologists are not in accord as to when accommodation is so relaxed as to permit accurate refraction. Shaad [6] has given the opinions of several observers as to just how much mydriasis is necessary to relax accommodation. The recommended procedures varied from the use of atropine sulfate for two or three weeks daily, as suggested by Thorington, to Howe's suggestion of relaxation of accommodation for several months. Parinaud and Javal advocated about a week's use of atropine, and Cords expressed the opinion that three to eight days' use of the mydriatic is sufficient. In addition, Javal suggested augmenting the atropine solution with 3 drops of cocaine hydrochloride solution at the time of examination.

To determine for my own satisfaction just how much mydriasis is necessary and also to ascertain the relative merits of atropine and homatropine, I examined 27 children with both drugs. On the first day each patient received four instillations of 1 per cent homatropine hydrobromide solution in each eye at fifteen minute intervals. The patients were studied with the retinoscope, and a manifest examination was then done. After this each patient was given a 1 per cent atropine sulfate solution and directed to instil one drop in each eye three times a day for a week. They were then examined retinoscopically and received a manifest examination each day for seven days, after which the results were compared.

Results.—Homatropine: Only 5 of the 27 children tested with homatropine had reliable refractions as compared with the successive seven day examinations with atropine. One of the children showed myopia, which was never encountered during the succeeding examinations with atropine. Verifying the findings of Bothman,[13] 5 children were noted to have a higher astigmatism under homatropine than was noted at any of their daily refractions with atropine. In 3 of the 27 cases, however, the astigmatic error was higher with atropine than with homatropine.

Atropine: In 24 of the 27 cases the refractions performed with atropine showed consistency from the fourth until the last, or seventh, day. In the 3 other cases they became reliable after the fifth day.

The procedure which I now follow is the use of atropine for four to five days before refraction is attempted. In prescribing glasses a reduction of 1 or 2 D. from the final examination is made as is found necessary in the individual case. When full correction is given and the patient complains of blurring, the use of atropine is continued until the spasm is relieved.

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13. Bothman, L.: Tr. Am. Acad. Ophth. **42**:165, 1937.

There is no reason why poor visual acuity even in an infant should be allowed to become worse. I have frequently prescribed glasses for a child as young as 1 year old. The children wear the glasses comfortably, even crying when they are removed.

In many cases the vision improves with glasses, and it is often found of value at the clinic to follow the advice of Marshall [14] and repeat refractions often. This should be done at intervals of six months to a year.

IDEAL TIME TO INSTITUTE ORTHOPTIC TREATMENT

In view of the fact that an accommodative squint will sometimes take as long as six months after refraction to correct itself, a question often brought up is: How long a period must elapse after refraction before orthoptic treatment is instituted? Opinions vary.[6] I personally feel that if there is no improvement after six weeks of wearing glasses training should be begun. Surely no harm can come from this procedure. Needless to state, if the child's eyes become straight by correct refraction alone, the time required for cure will surely be lessened if orthoptic treatment is given.

THE ORTHOPTIC CLINIC

To reach the greatest number of persons it was found expedient to limit the number of lessons in the majority of cases to two a week.[15] Private patients received only one lesson a week.

The child must be in fair health and of average intelligence and comprehension. There must be no retinal disease, and there should be good fixation. The best age at which to begin treatment is between 5 and 6 years, although younger children are often accepted. In suitable cases home work, such as stereoscopic and cheiroscopic exercise, is given. In such cases the instruction at the clinic is proportionately decreased, depending, of course, on the progress. No home work is given in cases of abnormal correspondence or if a "false macula" is present. Actual orthoptic treatment is not begun until the vision is 20/70. Lessons are usually limited to one-half hour.

On the average it takes between three and eight lessons to get enough information about a squint (as to angle, retinal correspondence, hyperphoria, etc.) to enable the ophthalmologist and the technician to plan the particular procedure necessary in the individual case.

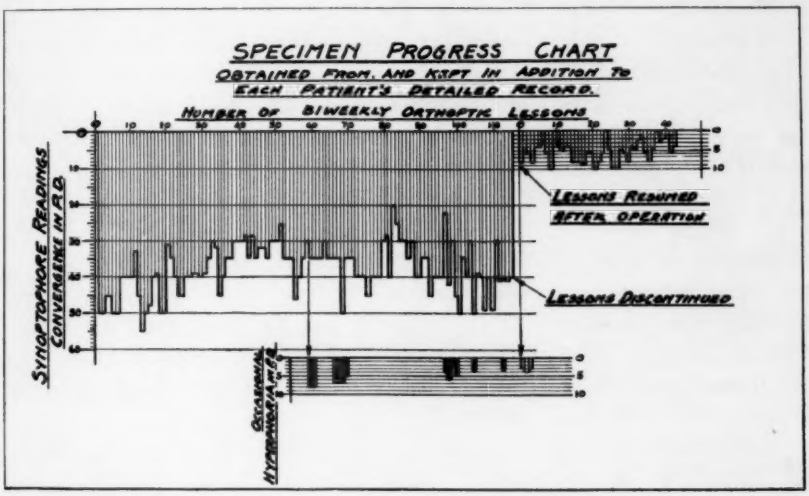
Orthoptic treatment is instituted postoperatively as soon as the dilated pupil has returned to normal size, provided there are no particular contraindications. If during the course of orthoptic treatment it is

14. Marshall, D.: Changes in Refraction Following Operation for Strabismus, Arch. Ophth. **15**:1020 (June) 1936.

15. Only 1 patient came once a week.

noticed after a reasonable time that there is no progress whatever, much time can be saved by advising immediate operation. A detailed record is kept of the progress made at each lesson; this, of course, would include the recording of any obstacle and the extent to which it is present. In addition to all this, I have found it helpful to plot the status of the squint by a chart constructed for the individual patient (reproduced here).

Instruments Used.—Various instruments are used at the clinic for measurement of the angle of squint and for classifying the various grades of binocular vision, but for training dependence is almost entirely on the synoptophore. It is essential in training, however, to maintain the



Progress chart of J. H., a girl aged 4 with vision of 20/30 in each eye. She took one lesson weekly. She had an alternating convergent squint of 50 prism diopters. Convergence improved to 30 prism diopters on the thirty-second lesson. A vacation of two months followed. It was noted that after cessation of treatment there was a return of the anomaly (eighty-third lesson). After the ninetieth lesson, however, fusion was permanently obtained. Progress stopped at the one hundred and tenth lesson, and the parents agreed to have the child operated on. The patient returned after operation (by Dr. A. G. Fewell) and obtained fusion with stereopsis on the thirtieth postoperative treatment. Occasional hyperphoria was noted throughout the course of treatment but at only three postoperative lessons. (This case is mentioned by permission of Dr. Fewell.)

child's interest, and for this purpose other instruments are sometimes found necessary. The stereo-orthopter (American Optical Co.), modeled after the Wheatstone instrument, or the telebinocular (Keystone View Co.), based on the Brewster apparatus, may be found useful.

Worth's classification of the three grades of vision is used. Grade 1 is subdivided into simultaneous macular perception with and simultaneous macular perception without superposition, and grade 2, into fusion with and fusion without amplitude. The Worth four dot test, the Maddox rod, anaglyphs and the simple experiment of the "hole in the hand" [16] are devices by which the three grades of binocular vision are determined. These also aid in the detection of suppression, hyperphoria, diplopia, etc.

HINDRANCES TO TRAINING

Obstacles to orthoptic training may be divided into two groups. In the first group are included such handicaps as suppression, amblyopia, vertical imbalance and abnormal retinal correspondence. In the second group there is absence of any of the grades of binocular vision.

Suppression.—Suppression is a disturbance in the visual function of an eye noted only when both eyes are used. Bielschowsky [17] claimed that suppression is brought on by loss of physiologic rivalry between the two eyes, a condition often observed in cases of squint. Suppression is commonly seen. Carleton and Madigan [18] observed an incidence of 71 per cent in their study of the horopter. Suppression may be present even though the patient has good stereopsis. It is believed to be the forerunner of amblyopia and may be associated with this condition in the same patient. Suppression makes it possible for abnormal correspondence to develop. At the clinic I had an interesting experience with 2 patients. In both the abnormal correspondence was broken up, but the suppression was not completely removed. Both returned within a year with early false projection.

One would think that the first point affected in a case of suppression would be be the macula. This, however, is not so. The area affected is a region corresponding to the visual direction of the fixing eye.

Evans,[19] using the Lloyd stereocampimeter, found a scotoma as small as 1 degree in some cases of squint in older children and young adults. Travers,[20] however, using the mirror screen test, found a somewhat larger suppression scotoma.

Diagnosis: The diagnosis of suppression is simple and can be made by any of the tests for binocular vision previously given. On the

16. Cantonnet, A.; Filliozat, J., and Fombeure, G.: Strabismus, translated by M. Coque, London, M. Wiseman & Co., 1934.

17. Bielschowsky, A.: The Physiology of Ocular Movements, in Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1940.

18. Carleton, E. H., and Madigan, L. F.: Size and Shape of Ocular Images: Clinical Significance, Arch. Ophth. **7**:720 (May) 1932.

19. Evans, J.: Am. J. Ophth. **12**:194, 1929.

20. Travers, T.: Brit. J. Ophth. **22**:577, 1938.

synoptophore the child does not notice the controls on the slide at the same time that fusion, or simultaneous macular perception, is obtained. Another test is not seeing the rosebud on the net in which the butterfly is later trapped. In a case of suppression the cheiroscope can also be used for diagnosis. The child is asked to put the ball, held in his hand by a small rod, into a small basket presenting itself before the other eye. The child with suppression invariably crosses the septum with the rod hand to the side of the basket.

Treatment: This consists in calling particular attention to the details of the slide, especially to the suppressed area. Simple pictures, such as the star in the circle, are useful. Failing with this, one may resort to any of the following devices: (*a*) flashing the light moderately, (*b*) increasing the intensity of the light over the target before the eye, (*c*) shaking or moving the slide, (*d*) when using the synoptophore, moving the arm which carries the slide or target, and (*e*) telling the patient to blink or pointing to the suppressed area with a colored indicator.

Red, yellow and blue have proved excellent for the treatment of suppression. It has been correctly stated [16] that colors are useful for this purpose because they demand attention and impress a larger area of the retina. In addition, fixation and accommodation are not necessary in looking at colors.

Home Work: Occlusion is sometimes employed. The patient must be reliable before stereoscopic or cheiroscopic exercises are used. The exercise of the "hole in the hand" is sometimes beneficial.

Results: I give the figures for the patients of the clinic and the private patients separately to show that it is possible for two different observers to publish widely different results when the same anomaly is investigated. For both groups the incidence of suppression was over 50 per cent [21] (table 1). The most easily cured patient required nine lessons, and the most stubborn condition required forty-two treatments.

Amblyopia.—This is a condition in which the vision is poor without visible pathologic change in the eye. It is possibly caused by suppression following an uncorrected error of refraction. Summerskill [22] expressed doubt whether any direct cause or secondary anatomic lesion is responsible for the condition. He stated the belief that blindness is evidence of a psychologic inhibition. Smukler [23] concluded after study of a number of cases that the cause of amblyopia is a retinal hemorrhage following birth and cited a number of observers of the same opinion.

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21. In the results given for the various obstacles only gross figures are used. See tables 1 and 2 for exact figures for each of the anomalies.

22. Summerskill, W. H.: Brit. M. J. **1**:1101, 1936.

23. Smukler, M. E.: Pennsylvania M. J. **37**:25, 1933.

Relation of the Angle of Squint to Amblyopia: Opinions differ as to the relation between the angle of squint and the degree of amblyopia. Zimmerman [24] expressed the belief that the angle of squint is usually larger with the higher amblyopias. This was observed among my patients in the clinic. Miranda,[25] however, found no relation between the angle of squint and amblyopia. This phenomenon was noted in reviewing my private cases. I cannot explain the radical difference in the findings for my two groups; it shows how correct data collected from different groups may differ. Amblyopia may of course be present without squint, but if the two conditions occur together it is impossible to say which is the cause and which the effect. I have often seen two or three squinting members of a family, all with amblyopia.

Fields: Haitz [26] cited Heine and Uhtoff as having found consecutively a 90 and 50 per cent incidence of scotoma in cases of unilateral

TABLE 1.—*Obstacles to Orthoptic Training Observed in Fifty-Six Private and Seventy-Two Clinical Cases*

Incidence			Cured	
Private Patients, per Cent	Clinic Patients, per Cent		Private Patients, per Cent	Clinic Patients, per Cent
Constant 74	54			
Occasional 6	6	..... Suppression	40	46*
42	55	..... Amblyopia	46	55*
Constant 60	32			
Occasional 20	23	..... Vertical imbalance	10	None
14	30	..... Abnormal retinal correspondence	50	50

* Improvement possibly due to the more frequent orthoptic training given patients in the clinic.

amblyopia. Haitz devised a binocular stereoscopic instrument by which he could outline a scotoma as small as 2 to 6 degrees and connected with the physiologic blindspot. Using the ordinary perimeter, I was not as successful with children. Each of 6 children with squint and unilateral amblyopia (vision 6/60) had a contracted field, and only 1 of these had a central scotoma. Each of 3 children with unilateral amblyopia (vision 6/21) had only moderately contracted fields. Against these children I checked 4 adults with unilateral amblyopia (vision 6/60). Only 1 had a markedly contracted field; 2 had normal peripheral fields with a central scotoma; the remaining 1 had a normal peripheral field with a temporal scotoma. Of 9 patients with unilateral amblyopia without squint, only 1 had a contracted field with a small central scotoma.

24. Zimmerman, H. E.: Klin. Monatsbl. f. Augenh. **100**:600, 1938.
25. Miranda, A. G.: Arch. de oftal. hispano-am. **35**:393, 1935.
26. Haitz, E.: Klin. Monatsbl. f. Augenh. **99**:761, 1937.

Cause: The direct cause of amblyopia is most likely a high refractive error, such as hyperopia, astigmatism of high degree, a combination of these or anisometropia.

Prognosis: Bressler [27] claimed that squint with uncured amblyopia will result in a 75 to 80 per cent cosmetic cure only. Children up to the age of 8 years respond best to treatment. One is not, however, so fortunate if treatment is instituted late in life. The prognosis is even poorer for older persons if there is a high visual error. Poor fixation and an associated anomaly, such as a vertical imbalance, make for a poor prognosis, in much the same way as would amblyopia complicated by a high degree of squint.

Treatment: An excellent method of training to correct deep amblyopia [16] is as follows: The hand is placed sagittally between the eyes of the patient. The amblyopic eye fixes a strong light, which may be reduced in intensity as the vision improves. Before the other eye a hand or a toy is moved in all directions. This method is also useful in the treatment of suppression.

Complete and constant occlusion of the good eye, with frequent visual checking of each eye, is also used. As was stated by Gifford,[28] it has been found that prolonged occlusion in the presence of the high amblyopias is next to impossible.

Another method is the placing of a deep red glass before the good eye. This may be used as a substitute for occlusion and has sometimes proved beneficial. The saturation of the colored glass may be gradually reduced as the condition improves. Atropine may be used in place of occlusion when the vision reaches 20/50. Visual training as described in a previous paper [1b] is often made use of.

The opinion is universal that orthoptic training should not be instituted until the vision is 20/70.

Results: It was noted that a number of these patients were anisometropic. It was also observed among the patients with amblyopia that improvement took place between the tenth and the twenty-fifth lesson. In other words, if the patient by using the treatment described did not improve in six months his amblyopia remained at a standstill.

Some improvements were so little short of miraculous that I almost hesitate to report them. However, these improvements were gradual, which confirms their authenticity. In the entire group about one half of the patients had amblyopia, and about one half of these improved (table 1).

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27. Bressler, J. D.: Treatment of Strabismus: Influence of Orthoptic Training on Results of Operation, Arch. Ophth. **16**:433 (Sept.) 1936.

28. Gifford, S. R.: Brit. J. Ophth. **19**:148, 1935.

Vertical Imbalance.—Vertical imbalance is a complication often associated with squint. White and Brown [29] claimed that 2 of every 5 patients with squint have a vertical imbalance, and they expressed the opinion that this condition is a factor in producing convergent and divergent squint. While this may be so, it was noted during orthoptic treatment that when the squint improved the vertical anomaly did not show a corresponding reduction. I have found that, as has been pointed out by Wells,[30] low grade hyperphoria may disappear as convergence power increases.

Lyle and Jackson [31] stated that postoperative hyperphoria improves in a few days (chart). Sometimes, however, it takes a much longer time. In cases of constant postoperative hyperphoria one wonders what role anatomically wrong fixation of the eye during a faulty operation has played in the anomaly.

Dunnington [32] expressed the belief that hyperphoria is paretic rather than concomitant even if the muscle action is concomitant. My results, particularly in the clinic cases (table 1), bear out this opinion. Dunnington claimed that for a correct diagnosis the muscle balance should be taken in the six cardinal directions.

Diagnosis: This is easily accomplished with the synoptophore, the maddox prism [33] or the heterophorometer.[34] In this series hyperphoria often did not present itself at first but appeared after a few lessons. An easy classification of this condition is that given by Cantonnet and Filliozat: [16]

(*a*) Slight vertical hyperphoria, a common condition and one which easily disappears.

(*b*) Easily noticeable hyperphoria, usually associated with lateral squint and following operation.

(*c*) True vertical hyperphoria, a rare type.

Results: The opinion is general that vertical imbalance is a formidable barrier in training for squint. This opinion has been expressed by a number of investigators, among whom are White and Brown [29]

29. White, J. W., and Brown, H. W.: Occurrence of Vertical Anomalies Associated with Convergent and Divergent Anomalies, Arch. Ophth. **21**:999 (June) 1939.

30. Wells, D. W.: The Stereoscope in Ophthalmology, Boston, E. F. Mahady Co., 1926.

31. Lyle, K., and Jackson, S.: Practical Orthoptics, London, H. K. Lewis & Co., Ltd., 1937.

32. Dunnington, J. H.: Am. J. Ophth. **14**:1140, 1931.

33. Dobson, M.: Binocular Vision and the Modern Treatment of Squint, New York, Oxford University Press, 1933.

34. Bielschowsky, A.: The Signs and Symptoms of Heterophoria, in Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1940.

and Davis [35] Hardy, however, in discussing a paper by Prangen,[36] expressed a more optimistic view of the treatment of hyperphoria as a complication. On the basis of results obtained at the clinic (table 1), where there were no improvements, and those obtained with my private patients, of whom only 10 per cent were cured, I consider this a grave complication in the orthoptic treatment of squint.

Abnormal Retinal Correspondence.—Abnormal retinal correspondence has been given its due credit as the great obstacle to orthoptic training by a long list of authors. It is a condition in which the patient uses the macula of one eye with the nonmacular area of the other eye. It is an acquired condition and is believed to develop before the age of 2 years and to decrease in frequency up to the age of 4 years. Opinions differ as to whether there is actually a point to point correspondence in the retinas. Henschen [37] seems to have located the center of retinal correspondence in the visual cortex. Burri [38] gave a résumé of the various theories proposed for retinal correspondence, questioned their rationale and suggested a theory of her own.

Diagnosis: The synoptophore is the only instrument relied on for both diagnosis and treatment. There is at the physician's disposal also the congruence apparatus of Tschermak and the after-image test popularized by Bielschowsky.[17] However, neither of these has proved satisfactory in the diagnosis or treatment of the condition, since children are not always dependable in subjective tests. Travers [20] correctly stated that retinal correspondence has to do with fusion. He expressed the belief that fusion of macular images is more reliably determined by the synoptophore and the mirror screen test than by those devices which measure the macular image of one eye and an eccentric point of the other eye, such as the maddox test or the test for diplopia.

My technicians have noticed even in the course of treatment that the patient wavered between normal and abnormal correspondence in the same lesson. This was most often seen during the stage of improvement.

Treatment: The synoptophore treatment of this condition is accomplished by one of two methods.[6] Technicians becoming adept at one form of treatment utilize this technic in all their subsequent cases. In this series the same method was used for all the private patients, in whom the two maculas were simultaneously stimulated at the objective angle of deviation with repeated lateral movements.

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35. Davis, W. T.: Evaluation of Orthoptic Training in the Treatment of Squint, Arch. Ophth. **14**:482 (Oct.) 1935.

36. Prangen, A. de H.: Relation of Orthoptic Training to the Surgical Treatment of Concomitant Strabismus, Arch. Ophth. **14**:683 (Oct.) 1935.

37. Henschen, S. E.: Arch. f. Ophth. **117**:403, 1926.

38. Burri, C.: Concept of Abnormal Retinal Correspondence: Theoretical Analysis, Arch. Ophth. **19**:409 (March) 1938.

On the other hand, it was definitely noticed at the clinic, where both methods were used, that certain persons lend themselves better to one form of treatment than to another. One should therefore select the method of treatment which best suits the individual patient. It is important to remember that improved patients should remain under care for several years, returning every six months for a recheck. At the clinic this was forcibly emphasized by the cases of 2 patients discharged a year previously who returned with abnormal correspondence. Fortunately this condition responded quickly to treatment.

Prognosis: It is best not to give treatment for a high angle of squint with abnormal correspondence. This encourages a "false macula" and adds to the pathologic process already present. Travers [3] expressed the belief that the "confusion" of von Kries is a temporary abnormal correspondence. He expressed agreement with Braun [8] that in some cases there is an innate weakness of retinal correspondence which accounts for the obstinacy of this condition. Correction of the anomaly before operation is preferable. Whenever possible I have found this to be much more beneficial than depending on operation alone for a physiologic cure. Hicks and Hosford [39] expressed the opinion that the condition is a hindrance to both operation and treatment. Travers [3] gave the opinion of several observers as to the prevalence of the condition. Prangen [36] cited Pugh as having stated that false projection runs high among alternators and that if more patients were intensively treated after operation the result would be surprisingly good. My experience, however, shows this anomaly to be difficult to treat, particularly in cases of the alternating type of squint, regardless of how intensive the treatment.

Results: Improvement in my group of cases occurred usually when the angle of the anomaly was from 10 to 15 degrees. The least number of lessons given to effect a cure was twenty. The majority of patients, however, took considerably more instruction. Some were treated for several years without relief, only to be cured by operation (chart). The incidence of abnormal correspondence varied in the two groups studied. The resultant cures were about 50 per cent (table 1).

Having overcome the aforementioned barriers to orthoptic training, one is ready to attempt to obtain the three grades of binocular vision. In the treatment of the aforementioned obstacles one is naturally training perfection in simultaneous macular perception with superposition of images. However, I am separating the three grades of binocular vision for the reason that when a patient has reached this stage stress is particularly laid on the grades of binocular vision with interspersing of some treatment to make sure that the previous obstacles are still under control.

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39. Hicks, A. M., and Hosford, G. N.: Orthoptic Treatment of Squint, Arch. Ophth. **13**:1026 (June) 1935.

SIMULTANEOUS MACULAR PERCEPTION; FUSION; STEREOPSIS

Before actual orthoptic training for the three grades of binocular vision is begun, the vision should be at least 20/70. As has been mentioned, suppression and abnormal retinal correspondence should be broken up before training for fusion is begun. Among my cases, even when all these obstacles had been overcome and fusion and stereopsis obtained, there remained 17 cases in which operation was necessary to obtain ocular parallelism. Needless to say, when operation is resorted to in cases of good binocular vision the results are satisfactory.

Simultaneous Macular Perception.—Diagnosis: This entity is possibly the simplest of the three grades of binocular vision to obtain. However, all patients do not respond easily to treatment. In this series there were some patients who never obtained simultaneous macular perception with the most intensive treatment.

Prognosis: In the cases in which simultaneous macular perception was obtained the minimum number of lessons was eight and the maximum thirty-two. Fewer patients among the private group lacked simultaneous macular perception, and a greater number of them than of the patients at the clinic showed improvement. It is possible that the treatment which they received prior to coming under my care was responsible for this. Over one half of the patients who did not have simultaneous macular perception originally acquired it with treatment.

Fusion.—The attainment of fusion is essential in overcoming a squint. Bielschowsky [17] stated that as long as the fusion apparatus is functioning there may be a temporary, but there is never a permanent, squint.

Diagnosis: A number of targets are available for use with the synoptophore or any of the stereoscopes. For the latter, the Keystone, Guibor and Sattler cards are useful. Since children tire easily of seeing the same pictures continuously, my technicians are always making their own targets. They are easy to make and inexpensive. This also applies to slides for simultaneous macular perception and stereopsis.

Treatment: During training for fusion one should always be on the alert to detect just when, if at all, suppression sets in. Permanency of fusion is assured by developing amplitude and by rechecks for many months after the cure is supposed to have been effected. During tests for amplitude of fusion a child may say that fusion is present long after the arms of the synoptophore are separated to the limit of fusion. On close questioning it develops that the child has not obtained fusion but is afraid to admit the fact. In this connection it is important to remember that children's answers bear close investigation. Children occasionally let their imaginations run away with them.

The simplest form of fusion is color fusion. Yellow or blue is suitable for this type of training. Anaglyphs in circles and V-shaped lines are used. These are viewed with a complementary colored glass before each eye.

Adduction and Abduction: For diagnosis as well as for treatment it should be remembered that the relation between adduction and abduction should be between two and four of adduction to one of abduction. This is more important than individual measurements. Exercises in adduction and abduction must be given with the greatest care to avoid fatigue. This is important to remember in the training of school children. They have had a full day at school and come to the clinic somewhat tired. For this reason it is felt that the ideal patient is the preschool child. Krimsky [40] mentioned a few factors which affect adduction and abduction, namely, worry, fatigue, illness, concentration, speed of targets and type of pictures. Exercises can be given effectively to children between the ages of 3 and 7 years. They are sometimes more easily accomplished with stereoscopic than with flat, and with simple than with complicated, pictures. It is well to remember that with convergent squint the purpose of treatment is to relax excessive convergence. A prism with the apex in creates an artificial divergence, thus bringing the object to the macula. In this case the object of the prism is to relax hyperfunction. With divergent squint, however, there is no hyperfunction but there is poor convergence. One should not use a prism, for then the patient will make no effort toward improvement. When there is need of a stimulant to mental effort the prism is useful. With improvement, of course, the prism is reduced. To get a divergent squint with normal binocular vision to converge still more, a prism apex in up to 20 D. should be used. An excellent exercise [16] for practice at home is to imagine an airplane in the distance going down gradually and landing at one's feet and then to follow the takeoff of the plane as it goes into the extreme distance. This makes use of the fact that divergence is greater in the upper field and convergence is correspondingly greater in the lower field.

Results: The smallest number of lessons required to obtain fusion was seven and the largest, forty-one. Seven lessons sufficed in only a few cases. Less than half of the patients obtained fusion (table 2). One intelligent and cooperative patient did not obtain fusion even in one hundred and fifteen lessons.

Stereopsis.—Stereopsis is the third and highest grade of binocular vision in the classification of Worth. As was noted previously, in discussing other obstacles, it is always best in treating this condition

40. Krimsky, E.: Psychologic Considerations in Study of Binocular Function, Arch. Ophth. **21**:662 (April) 1939.

to choose simple pictures, preferably in color. Stereopsis should be differentiated from perspective, which is not actually the third dimension but the artist's conception of space. This the artist easily accomplishes by a difference in size of the various objects he paints, in association with lights and shadows. It is sometimes difficult to explain to the child just what is meant by stereopsis, so that in the beginning one questions whether the child is actually successful in its attainment.

Results: For those who eventually did obtain stereopsis between eleven and eighty-five lessons were required. A little over a quarter of the patients who had no stereopsis originally were successful in attaining it (table 2).

TABLE 2.—*Absence of Indicated Functions in the Individual Grades of Binocular Vision*

Incidence			Cured	
	Private Patients, per Cent	Clinic Patients, per Cent	Private Patients, per Cent	Clinic Patients, per Cent
Constant	35†	45		
Occasional	10	15	Absence of simultaneous macular perception 87†	56
Constant	25	49		
Occasional	31	28	..... Absence of fusion..... 36	49*
Occasional	None	7		
Constant	37	55	..... Absence of stereopsis 29	38*

The patients at the clinic received two to three lessons weekly; the private patients, one lesson a week.

* Improvement possibly due to the more frequent orthoptic training given patients in the clinic.

† The low incidence of lack of simultaneous macular perception for the private patients and the high degree of improvement are due to training received prior to my acceptance of these patients.

ANISEIKONIA

Much has been said in recent years of this serious anomaly, which may play a part in the role of causation of squint, particularly alternating squint. At present, however, the technic is somewhat complicated, time consuming and expensive. The use of the Andrews card and a similar device has been attempted for gross segregation of patients with this condition. As yet the results are conflicting and do not lend themselves to valid reporting.

PSYCHOLOGIC EXAMINATION

In a desire to find a neuropsychologic basis for some of the most stubborn conditions observed in patients of the clinic, I selected the 6 children who responded least to orthoptic treatment. Each of the children had been operated on from one to three times either at this hospital or at another. All had squints of the alternating type. Each underwent a complete examination in the various departments of the hospital, followed by examination by the staff psychologist, who investigated the personality as well as the emotional and intellectual factors.

All the children had average or slightly superior ability, with normal personalities. They varied in specific ways which might influence the success of their training. Table 3 is an abbreviated report of the pertinent findings.

Since this report was obtained several of these children have been operated on (tenotomy), with a good cosmetic result. It is unfortunate that I did not have 6 orthoptically cured patients examined in like manner. These would act as controls, even though the number for comparison were small. However, in the face of the discouraging reports received in these cases of stubborn strabismus I am inclined to believe that in a case of squint, particularly of the alternating type, it is advisable to make a complete psychologic examination before attempting orthoptic treatment. This might obviate spending two or three years

TABLE 3.—*Results of Neuropsychologic Study*

Patient	Sex	Age	Comment
1. B. G.	F	10	Underweight; poor muscular tone
2. E. A.	F	6	Defect in memory span and development of memory concepts; immature in appreciation; sought attention by failing to respond
3. J. B.	M	10	Malnourished; showed deficiencies in attentional persistence (superficial attention) but with superior appreciation of spatial relations
4. G. L.	M	7	Extremely unstable environment reflected in his own restlessness; emotion, lack of concentration, attention and resistance to authority; slight deficiency in eye-hand coordination
5. L. S.	F	13	Questionable motivation of any kind of learning
6. J. K.	M	9	Malnourished; immature; defect in discrimination of perception and lack of comprehension of directions; dislike for glasses and training; lack of persistence

treating a condition with which one could obtain the same cosmetic result in much less time by surgical intervention.

REVIEW OF THE CLINIC CASES

It is impossible to follow up private patients. Many of these were referred under the care of their own ophthalmologists. I have, however, tabulated the two groups of patients separately as to incidence of the anomaly and percentage of patients ultimately cured. This was for the purpose of showing how two separate investigations may result in different ratios for the same condition. A review of the data on the patients of the clinic merits comment here. Of the total number of 72 patients, 38 had alternating squint, and 34, unilateral squint. Sixteen of those with unilateral squint were dropped from my list, although a few were ready for discharge. They either moved to a great distance or showed lack of cooperation (poor attendance). By orthoptic training 5 patients with alternating and 8 with unilateral squint obtained normal physiologic function with ocular parallelism.

For alternating squint the shortest orthoptic training period was about two months, and only 1 patient ever got well in so short a period. Most of the patients with alternating squint took several years. The patient with the most stubborn alternating squint which was cured required two and one-half years. Both these patients had a 10 degree squint (table 4). With unilateral squint (table 4) the shortest period required for cure was about four and one-half months (for an 8 degree squint), and the longest, seven months (for 25 degree squint). It should be repeated that while the duration of treatment of unilateral squint was up to seven months, the successfully treated squints were usually no higher than 15 degrees.

TABLE 4.—*Four Extremes Cured by Orthoptic Treatment*

Patient	Sex	Age	Vision	Comment
				Alternating Squint (10 Degrees in Both Cases)
K. S.	F	11	20/30 20/20	Eight pretreatment examinations given to determine alternation; occasional hyperphoria of 2 prism diopters; fusion obtained at first lesson; alternating suppression relieved in 6 lessons; discharged after thirteen weeks; lessons biweekly for about 2 months
G. A.	M	8	20/20 20/20	Operated on 3 months prior to admission; abnormal correspondence; discharged after 119 lessons; only clinic patient to come but once a week (lived great distance from hospital)
				Unilateral Squint
D. K.	M	8	20/40 20/40	Convergent squint of left eye, 7 degrees; discharged after 30 biweekly lessons; vision improved to 20/30 in each eye
M. E.	F	9	20/100 20/ 20	Convergent squint of right eye, 25 degrees; simultaneous macular perception in 13 lessons; fusion in 21 lessons; discharged after 52 treatments with vision 20/30 and 20/20 (biweekly lessons)

SUMMARY

A report of the cases of 56 private patients and 72 clinic patients treated orthoptically is given under two separate headings. The variation in the data on the two groups for any of the anomalies will possibly explain how it happens that two observers arrive at somewhat different conclusions for the same entity. It also indicates that the two or three lessons a week given the patients at the clinic resulted more favorably than the single weekly lessons given the private patients. The predisposing factors and the phenomena usually thought to accompany squint are discussed. The requirements preliminary to orthoptic treatment are outlined. The superiority of atropine to homatropine for mydriasis is shown. The obstacles to orthoptic training are discussed, with their incidence in the groups examined and the percentage of patients finally cured of each particular anomaly. Perimetric studies of amblyopia, alone or combined with squint, are given for a few cases,

in which uniocular fixation was used. A chart used in addition to the detailed history is shown, by which the ophthalmologist can quickly note the cosmetic effect of the treatment.

Finally, 6 children with stubborn squint were examined by the neuropsychologic department. If the report had been received prior to the inauguration of orthoptic treatment it would possibly have affected my decision as to whether or not such a tedious and time-consuming procedure was worth while in some cases. Orthoptic treatment is of value in that it may often overcome an obstacle not remedied by surgical means. It supplements normal physiologic function with cosmetic cure when possible.

Blanche Rackow, of the social service department; Robert L. Schaeffer, of the neuropsychologic department; Annabel F. Taylor, in charge of the orthoptic clinic, and Dr. Frank Levy assisted in this study.

37 South Twentieth Street.

BOECK'S SARCOID

A CASE OF BILATERAL TUMOR OF THE LACRIMAL GLAND

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The case described here is apparently the second case of bilateral sarcoid of the lacrimal gland in which the patient underwent operative removal of the tumor.

On Nov. 14, 1939, one of us (M. M. C.) was consulted by E. M., a Negress aged 24, for a peculiar condition of the eyes. She presented a large swelling of each upper lid. She could see only by tilting her head backward at an awkward angle, as the openings between her lids were narrow slits. The bilateral swelling had been first noticed in July 1939. The tumors had gradually increased in size since that time. She had been seen by a number of physicians, none of them ophthalmologists, who had been frankly puzzled and had told her so. The condition was diagnosed as bilateral tumor of the lacrimal gland. There was a peculiar associated condition. The turbinate bones on both sides were hypertrophied to such an extent that it was impossible for her to breathe through her nose. It was hardly possible to force a probe between the turbinates and the septum. There was a harassing racking cough that kept her awake at night. She had steadily lost weight to about 85 pounds (38.6 Kg.). Her normal weight was 140 pounds (63.5 Kg.). A general physical examination was ordered. The Wassermann reaction was negative. Examination of the chest gave negative results for tuberculosis. The chest and the sinuses were roentgenographically normal. A therapeutic test (mixed treatment with small doses of mercury bichloride and potassium iodide) was given, which seemed to improve her physical condition. Removal of the tumors was advised. The tumor on the right eye was removed first, as it was the larger. Operation was done on January 23 with the patient under general anesthesia. The operation presented considerable difficulties, as this type of tumor slides around and has a way of disappearing under the brow. Then, too, it is somewhat difficult at times to differentiate the tumor from the orbital fat. The incision was made in the eyebrow to conceal the scar as much as possible. The tumor was a much hypertrophied lacrimal gland. It was sent to the laboratory and was reported as carcinoma. This was, of course, disturbing. A reexamination was requested, and this time a diagnosis of tuberculosis was made. The slides were then submitted to one of us (E. W. G.), and the condition was promptly identified as a rare form of tumor generally known as Boeck's sarcoid. It was described as an infectious process and not malignant. It was thought important to examine the other specimen; so the patient was placed in

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the Vanderbilt University Hospital, and the tumor was removed from the left eye on April 11. The same condition was found in the tumor from the left eye.

Pathologic Report (E. W. G.).—Microscopic sections of the lacrimal glands showed a diffuse granulomatous process characterized by multiple miliary lesions resembling, and in fact histologically indistinguishable from, proliferative tubercles. There was no caseation. Giant cells of the Langerhans type were numerous. No tubercle bacilli were seen, and animals inoculated with ground material from the lesions did not have tuberculosis. The lesion is characteristic both of Boeck's sarcoid and of uveoparotid fever, which from clinical considerations Longcope and Pierson [1] suggested are expressions of the same infectious process. This view was also expressed by Cotter,[2] because of similar cardiac lesions that have been found in both conditions.

Fig. 1.—Photograph of the patient's eyes.

Pathologic Description in Literature.—The following description was given by Cotter:

"Clinically, sarcoid is a chronic disease. It persists usually over a number of years, produces only a slight constitutional reaction, seldom responds to treatment, is sometimes recurrent, and most often is associated with a negative tuberculin reaction. The clinical manifestations are characteristic lesions of the skin, enlargement of the superficial lymph nodes and tonsils, widening of the hilar shadows of the lungs, as observed roentgenographically, infiltration of the peripheral fields of the lungs, rhinitis, iridocyclitis, enlargement of the parotid gland, splenomegaly and hepatomegaly. Apparently the tissues of different persons vary markedly in susceptibility. In one case the skin may be predominantly involved; in another, the

1. Longcope, W. T., and Pierson, J. W.: Boeck's Sarcoid (Sarcoidosis), Bull. Johns Hopkins Hosp. **60**:223, 1937.

2. Cotter, E. F.: Boeck's Sarcoid: Autopsy in a Case with Visceral Lesions. Arch. Int. Med. **64**:286 (Aug.) 1939.

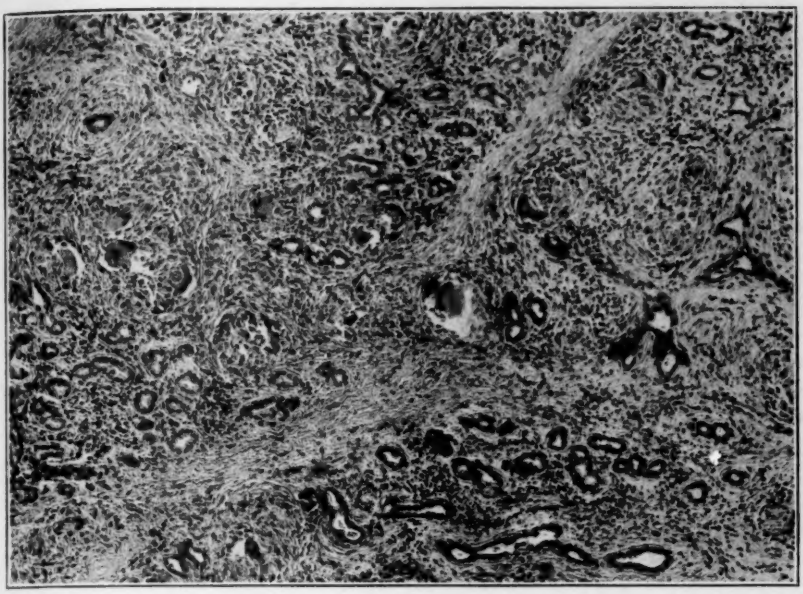

Fig. 2.—Low power photomicrograph of the lacrimal gland, showing the diffuse granulomatous lesion. × 120.

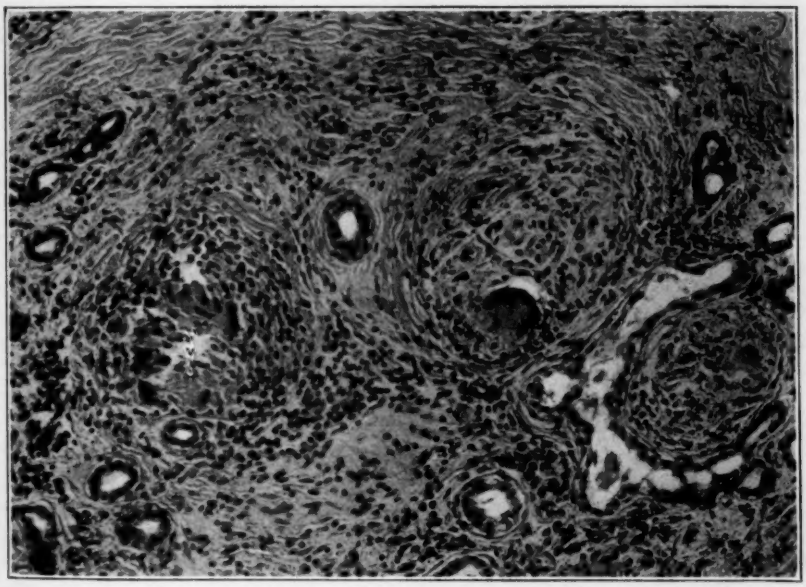

Fig. 3.—Miliary lesions resembling proliferative tubercles. × 225.

lymph nodes, and in still another, the internal organs. Cutaneous lesions are not present in some cases.

"Longcope and Pierson reported a case of Boeck's sarcoid with bilateral iridocyclitis in which also the superficial lymph nodes were enlarged. The lacrimal glands were swollen, and nodules appeared beneath the eyelids and in the parotid glands. Roentgen examination of the chest revealed an enlargement of the hilar shadows and an infiltration of the lungs, particularly on the right side. The authors called attention to the striking resemblance between the condition and that described as uveoparotitis, uveoparotid tuberculosis or uveoparotid fever. Uveoparotid fever presents the same etiologic problem as sarcoid. It is a chronic condition in which there is mild fever, bilateral uveitis and enlargement of the parotid glands. The disease of the uveal tract usually leads to some impairment of vision, while the parotid glands return to their normal size. Not infrequently there is paralysis of the facial nerve. The tuberculin test is usually negative except in the presence of an active classic tuberculous lesion. Typical cutaneous lesions and osseous changes of sarcoid have not been described as associated with the disease."

Course.—The removal of the tumors was followed by a surprising effect on the patient's physical condition. The hypertrophied turbinates rapidly returned to normal, and the patient breathed easily through the nose. The distressing cough disappeared completely. The patient at once began to put on weight, and at the time of writing, almost a year after the first operation, she has gained 75 pounds (34 Kg.) and weighs 160 pounds (72.6 Kg.). The most interesting feature of the case is the striking improvement in the patient's physical condition following the operations. The explanation would seem to be that the infectious material forming in the glands passed into the eyes and from there by way of the lacrimal ducts into the nose; from there it followed the airway into the larynx and the trachea. The infection in the nose gave rise to hypertrophy of the turbinates, and the infectious material draining into the larynx and trachea gave rise to the severe cough. When the source of the material was cut off the mucous membrane in the nose and trachea rapidly returned to normal.

Cases of tumor of the lacrimal gland are extremely rare. In a world-wide search of the literature covering a period of three hundred and twenty-three years Lane found only 223. One of us (M. M. C.) has reported 1 previous case, in which a large tumor of the gland had the characteristics of a sebaceous cyst. It was successfully removed.

The types of tumors of the lacrimal gland are: adenoma, adenocarcinoma, angioma, chrondroma, dacryops, cyst of the accessory gland, dermoid cyst, endothelioma, epithelioma, granuloma, lymphoma, Mickulicz' disease, mixed tumor, adenosarcoma, adenomyxoma, lymphangioma, sarcoma, mixed sarcoma and tuberculoma.

Our case of bilateral sarcoid of the lacrimal gland appears to be the only case ever reported in which operation was performed.

The lesson to be learned appears to be that when the disease is localized surgical removal is indicated if possible.

A similarity may be noted between the case presented here and that reported by Longcope and Pierson, in which there were enlargement of the lacrimal glands associated with bilateral iridocyclitis and enlargement of the superficial lymph nodes. Nodules appeared beneath the eyelids and in the parotid glands. Roentgen examination of the chest revealed enlargement of the hilar shadows and infiltration of the lungs, particularly on the right side.

APLASIA OF THE OPTIC NERVE

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Aplasia of the optic nerve is a congenital anomaly rarely seen except in grossly malformed eyes. Cords [1] (1923) said that it is one of the greatest rarities.

A brief review of the embryology of the optic nerve will facilitate understanding the condition.[2] By the 2.6 mm. stage of the embryo (fig. 1) the optic pits are becoming visible on each side of the anterior end of the neural canal. These pits rapidly grow out, forming by the 4 mm. stage the hollow, bulbous optic vesicles, the cavities of which are connected with the lumen of the neural canal by the short optic stalks. At the 4.5 mm. stage (fig. 2) the fetal fissure begins to form by invagination of the optic vesicles in a ventrolateral position. The outer wall of each vesicle, which is to become the retina, has by this time increased to several cell layers in thickness and is in contact with the lens plate of the surface ectoderm. At the 5.5 mm. stage (fig. 3), through further invagination of the fetal fissure, these cells have nearly come in contact with the posterior layer of the optic vesicle, which is later to become the pigment epithelial layer of the retina. They are separated from the lens plate by mesodermal tissue, which has grown along the groove in the optic stalk and through the fetal fissure. At this stage the fetal fissure extends from the ventrolateral portion of the margin of the optic cup into the optic stalk. The point where the fissure ends on the stalk marks the point of future entrance into the optic nerve of the central retinal vessels, which are in the process of being formed from the paraxial mesoderm that has invaded this groove and filled the

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Read before the Section on Ophthalmology, College of Physicians, Philadelphia, Feb. 15, 1940. The discussion which followed was published with the transactions of the Society (ARCH. OPHTH. **23**:1338 [June] 1940).

1. Cords, R.: Einseitige Kleinheit der Papille, Klin. Monatsbl. f. Augenh. **71**:414-418, 1923.

2. Mann, I. C.: The Development of the Human Eye, London, Cambridge University Press, 1928.

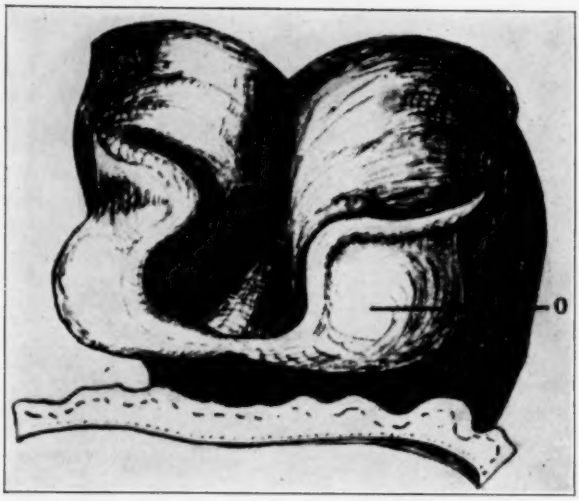

Fig. 1.—Model of the anterior end of the guinea pig embryo, the left optic pit being deep to the protrusion at *O*. (Figures 1 to 4 inclusive are reproduced from Mann, I.: The Development of the Human Eye, New York, The Macmillan Company, 1928.)

Fig. 2.—Front view of model of half of the forebrain and the right optic vesicle of a 4.5 mm. human embryo. The vesicle is cut open so that its cavity can be seen. Invagination of the vesicle is beginning.

optic cup. At this stage of embryonic life they are represented by the hyaloid system of vessels running through the upper part of the fetal fissure, at the site of the future optic nerve head, to the posterior surface of the lens. Until the 10 mm. stage is reached the only prominent changes are elongation of the optic stalk and continuation of invagination of the cup. Then the fetal fissure begins to fuse as its margins approach each other, first in the midzone and then extending distally to the margin of the cup and proximally. The proximal end of the fissure, or that at the posterior pole, cannot entirely close, for here the hyaloid vessels and their mesodermal sheaths have grown into the optic cup

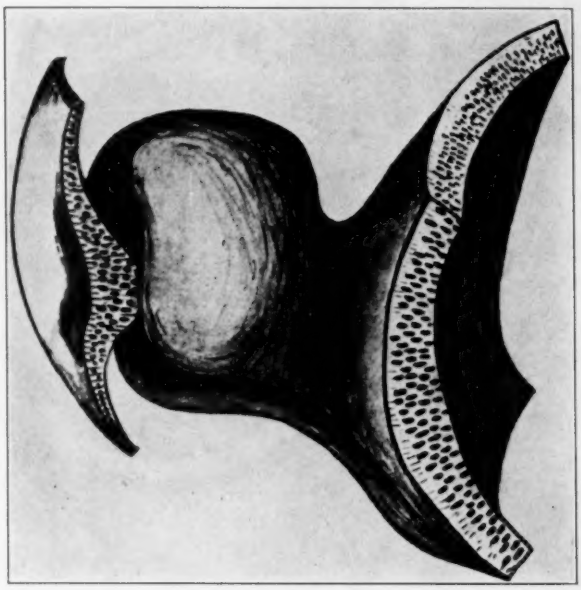

Fig. 3.—Model of the optic cup of a 5.5 mm. human embryo, showing invagination of the optic vesicle to form the optic cup.

and have formed the primitive papilla. It is conceivable that if the mesoderm and the vessels had previously failed to grow into the fetal fissure posteriorly, that structure might now close completely, leaving no optic nerve. Growth continues without striking change until the 17 mm. stage (fig. 4), at which time the retinal ganglion cells have appeared. They send off dendrites which enter the primitive papilla already containing the hyaloid vessels to contribute the neural elements of the nerve head. They then pass out through the papilla and the future optic foramen into the groove in the optic stalk and along its under surface toward the brain, forming the neural elements of the

optic nerve. They reach the chiasm at the 18 mm. stage and the tracts at the 25 mm. stage. As the nerve fibers grow along the stalk the cells constituting it are invaded, losing their identity, to form the glial cells of the optic nerve. The optic nerve sheath and the septums connected with this, as well as the central vessels and their sheaths, arise from paraxial mesoderm.

Consideration of the foregoing points would suggest that aplasia of the optic nerve might arise from failure of development of either the mesodermal elements, which supply the connective tissue and hyaloid vessels, or the ganglion cells of the retina. If the former failed to

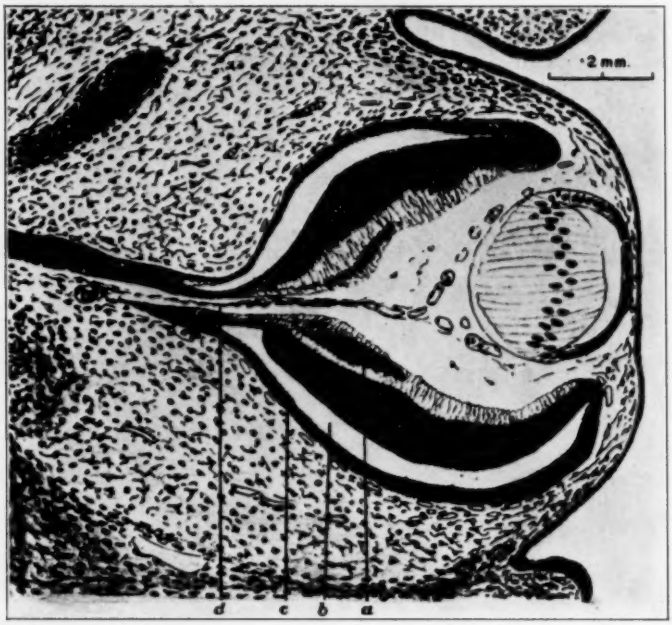

Fig. 4.—Section through the optic cup and stalk of a 17 mm. human embryo. At *a* is seen the inner layer of the cup with the inner neuroblastic layer; at *b*, the cavity of primary optic vesicle; at *c*, the outer pigmented wall of the cup, and at *d*, a hyaloid artery in the upper end of the fetal fissure.

develop, theoretically complete absence, or true aplasia, of the optic nerve with absence of the papilla might be predicted.

This is given support from several sources. Zeeman and Tumbelaka [3] in 1916 reported histologic studies of a congenitally blind cat in which no optic nerve or nerve sheaths could be found. No opening for the optic nerve could be found in the sclera, the chorioid or the pigment

3. Zeeman, W. P. C., and Tumbelaka, R.: Das zentrale und periphere optische System bei einer kongenital blinden Katze, Arch. f. Ophth. **91**:241-263, 1916.

layer of the retina. No central vessels were present. No ganglion cells, optic nerve fibers, optic chiasm or tracts were present. This careful study pointed to complete closure of the fetal fissure with no evidence of even a rudimentary papilla. Rosenbaum [4] cited Seiler, who reported examination of an eye from a patient with congenital hydrocephalus in which neither ganglion cells nor an optic nerve sheath could be found. In these cases it is reasonable to assume that both the mesoderm and the ganglion cells were aplastic. This type of defect has been seen only in grossly malformed eyes, as might be expected, since the mesoderm is important in the development of the eye.

Complete aplasia of the optic nerve has been seen ophthalmoscopically. Krauss [5] in 1920 saw a patient with myopia of 14 D. due to ectasia in the region of the entrance of the optic nerve, but neither the papilla nor the vessel funnel was visible. Retze in 1896 (cited by Cords [1]) reported a case of bilateral microphthalmos in which the right eye showed no evidence of an optic nerve; however, inferior to its expected site extensive spots of choroidal pigment could be seen. The papilla of the left eye was very large, slate gray, sharply bordered and surrounded by a white area having the appearance of a staphyloma. A small bundle of retinal vessels came out above and branched in the upper half of the fundus. From the description, the right eye of this patient and that of Krauss's patient would seem to represent a similar condition in a microphthalmic eye.

Three cases have been reported of complete bilateral congenital absence of the optic nerve in otherwise normal eyes, but analysis of the descriptions throws question on their authenticity. Brière [6] in 1877 saw a 7 year old girl, blind since birth, in whom the disk could not be seen; instead, the choroid was normal and uniform over the expected site. It was, however, traversed at that point by the normal central retinal vessels. As will be mentioned later, this would be more correctly called hypoplasia of the optic nerve due to failure of the ganglion cell layer of the retina, for the central vessels were present. Beard [7] in 1901 added to the literature a report on an 11 year old girl who was said to show absence of the optic nerves, but her case cannot be included, for she was not blind and her condition was probably acquired. She had suffered recent dizziness, emotional instability and vomiting. The disk borders could not be seen, and Beard stated that the appearance

4. Rosenbaum, S.: Beiträge zur Aplasie des Nervus opticus, Ztschr. f. Augenh. **7**:200-213, 1902.

5. Krauss: Kongenitale hochgradige Veränderungen an der Sehnerveneintrittsstelle, Klin. Monatsbl. f. Augenh. **64**:125-126, 1920.

6. Brière: Absence des papilles, cécité absolue, Ann. d'ocul. **78**:41-42, 1877.

7. Beard, C. H.: Congenital Absence of the Optic Discs with Condition Simulating Choked Disc, Ophth. Rec. **10**:281-283, 1901.

was that of recent papilledema. The central retinal vessels were present also. Later, one knee jerk was absent and the Romberg sign was positive. Her vision was 20/70 in the right eye and 20/50 in the left with correction. She was seen seven years later, and the fundi and the vision were unchanged. The history, the neurologic picture and the fundus picture were all compatible with increased intracranial pressure, and it seems that the diagnosis of absence of the optic nerves was made on the basis of obscured disk margins. Hawley [8] in 1911 also reported a doubtful case of absence of the optic nerves. The patient was a 12 year old girl; she was not blind but had failing vision of five years' duration. The fundus was described as normal except for absence of the disk outlines. Dr. Brown Pusey, in discussion of the case, said that he thought he could see the outlines of the nerve head in the right eye. This, with the history of failing vision, suggests an acquired condition with obliteration of the disk outlines. Thus none of these conditions can correctly be called congenital absence of the optic nerve, since in all of them the mesodermal elements of this structure were present. Brière's case, however, fits into the group about to be discussed.

If the mesoderm developed normally but the neural elements failed because of lack of development of the ganglion cells, a very small disk with central retinal vessels would be expected. Rosenbaum [4] in 1902 made histologic studies on the eyes of an anencephalic monster in which the optic disks were microscopically visible. The scleral foramen was filled with connective tissue, and the central vessels were present, with a persistent hyaloid artery in one eye. The nerve fiber and ganglion cell layer was absent from the retina. The nerve sheaths were present back of the eye, the greater part ending in the sclera, but the glia and central vessels continued into the globe. He also cited a case of microphthalmos reported by Dötsch in 1899, with a similar picture. Szymanski [9] in 1926 observed an optic disk of a cat which was deeply cupped and from which emerged small retinal vessels containing little blood. In this instance also the ganglion cell and nerve fiber layer was absent. This type of anomaly could be properly called aplasia of the ganglion cell layer of the retina or hypoplasia of the optic nerve and papilla.

This type of hypoplasia of the optic nerve has been seen ophthalmoscopically in externally well developed eyes with other congenital deformities. Krauss [10] in 1913 reported a case of unilateral aplasia of

8. Hawley, C. W.: Congenital Absence of Optic Disk, Ophth. Rec. **20**:27-28, 1911.

9. Szymanski: Les altérations rétiniennes dans l'oeil d'un chat privé de papille, Bull. Soc. franç. d'opht. **39**:265-272, 1926.

10. Krauss: Demonstration eines sehr seltenen Augenhintergrundbefundes mit Verlagerung der Durchtrittsstelle der Zentralgefässe, Ber. u. d. Versamml. d. ophth. Gesellsch. **12**:380-382 1913.

the optic nerve in such an eye in which the vision was limited to hand movements, with an absolute visual field defect present inferiorly and temporally. Strands of tissue ran from the pale disk to the retina above, where they were attached and whence finger-like processes projected into the vitreous. The retina seemed folded above. The retinal vessels came out from the white tissue above the disk. The other eye was normal. Magnus [11] in 1884 described an eye from which a congenital cataract had been removed, enabling view of the papilla, which was represented by a narrow white sickle bordered temporally by a brown pigment ring. Temporal to this was a grayish red area which was the place of emergence of the retinal vessels. No vessels ended on the white rudiment. Eversbusch (cited by Cords [1]) saw a patient in whom the entire posterior pole of one eye, except the macular region, was filled with medullated nerve fibers and in which the round form of the papilla could not be recognized. The vision was limited to counting fingers at 2 to 3 meters. From the description the nature of the anomaly seems uncertain. Duane [12] in 1905 saw a 2½ year old boy who was blind in both eyes. External examination revealed no abnormality except fixed pupils. A large opacity was present in the vitreous of the left eye. The disk was irregular, dirty and oval. The central vessels were present in each eye but could not be traced off the disk in the left eye and only a short distance farther in the right eye, being very fine. The macula in each was covered by a round area several disk diameters across, bordered by a sharp pigment ring. The area was pink but lighter in color than the rest of the fundus and contained many brown pigment spots. No difference in level from the rest of the fundus could be seen, and refraction showed 0.5 D. of myopia in each eye.

A few cases of hypoplasia of the optic nerve in eyes otherwise normal except for the appearance of the disk have been observed. Brière's case has already been mentioned. Cords [1] in 1923 wrote of a case of unilateral hypoplasia with convergent strabismus of the left eye of 10 degrees. The vision was 5/10 in the right eye, with a — 1.25 D. cylinder horizontally, and was limited to hand movements in the periphery of the left eye. The refraction of the left eye was — 1.25 D. in the vertical meridian. The pupils of both eyes reacted normally. The media were clear. The right disk was normal. The left disk was pale, the temporal half being almost white. The edges were well defined. With an F-50 ocular in the Gullstrand instrument, which gives a magnification of 9,

11. Magnus, H.: Zur Casuistik der angeborenen Sehnerven- Missbildungen, Klin. Monatsbl. f. Augenh. **22**:85-87, 1884.

12. Duane, A.: Aplasie der Papille und Netzhautgefässe met einer eigenthümlichen Anomalie in der Macula bei sonst normalen Augen, Arch. f. Augenh. **52**:351-352, 1905.

the vertical and horizontal diameters "were 20 and 19 respectively in the right and 13 and 12.5 mm. respectively in the left," which was about three-fifths normal for the latter. Roentgenograms of the optic foramen were normal. Velhagen [13] in 1930 contributed another case of unilateral hypoplasia accompanied with strabismus. The vision of the right eye was questionable for hand movements temporally; that in the left eye was 6/6. The disk of the right eye was only one-ninth to one-tenth the size of the left as measured with the Gullstrand instrument. The right disk was normal in appearance, seemingly a miniature of the left. The nasal two thirds had a pigment border. Temporally there was an area in which no blood vessels were visible, differentiated from the rest of the fundus by a grayish brown, lightly pigmented tissue. This area combined with a small disk made up an outline of what seemed to have been destined to receive a normal optic nerve, for it seemed the proper size. Refraction gave the same results for the two eyes. The right optic foramen was 1 mm. smaller than the left.

Schwarz [14] in 1915 observed an 18 year old girl with 6/12 vision in each eye, requiring correction (+ 0.50 D. cyl., axis 90) in the left. The visual fields showed almost complete binasal hemianopia. The color fields were proportional. The right disk was vertically oval and yellowish pink, being about half the normal width and three-fourths the normal height. A glassy cone projected inferiorly, possibly a remnant of a hyaloid vessel. Temporally and above was a yellowish red area edged with the fine pigmentation sometimes seen on the temporal side of a disk. The fovea was suggested by a darker area with the trace of a ring reflex. The left disk was about the same size but was round. The fovea could not be seen. The papillary vessels seemed to surround the disk. The condition was attributed to failure of development of the uncrossed nerve fibers. Ridley [15] in 1938 reported another case of the bilateral condition, the patient being a year old blind child. The pupils were fixed. The media were clear. The red reflexes from the fundi were pale, especially in the periphery. The choroidal pattern was well marked. Instead of normal optic disks there were excavations about 5 D. deep. They were gray. From the bases of the pits emerged only threads representing retinal vessels; the lower branch in the right eye was the only one which contained more than a trace of blood, and even this seemed to fade out soon after leaving the edge of the excavation.

13. Velhagen, C.: Ein Fall von einseitiger Hypoplasie des Sehnerven in einer sonst normalen Augapfel, Arch. f. Augenh. **102**:382-386, 1930.

14. Schwarz, O.: Ein Fall mangelhafter Bildung (Hypoplasie) beider Sehnerven, Arch. f. Ophth. **90**:326-328, 1915.

15. Ridley, H.: Aplasia of the Optic Nerves. Brit. J. Ophth. **22**:669-671. 1938.

REPORT OF A CASE

E. S., a 3 year old white boy, was admitted to the Hospital of the University of Pennsylvania on Oct. 3, 1939, having been referred by Dr. A. M. Ornsteen. His mother stated that he had been blind since birth. He had never suffered from any ocular disease. His general health had been excellent, with no childhood diseases. The family history revealed no similar condition and no neurologic disease. His brother was 6 years of age and was entirely normal and healthy. The patient was a full term child, delivered spontaneously. His gain in weight and general physical development were normal. His mother believed him to be precocious, stating that he seldom forgot a thing once learned. General physical examination

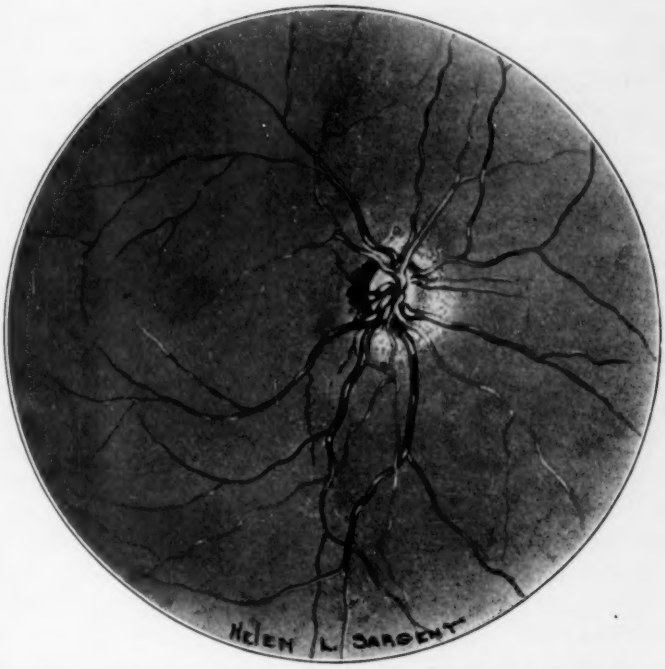

Fig. 5.—Photograph of a painting of the fundus of the right eye.

revealed no abnormality. Neurologic examination by Dr. Ornsteen revealed nothing except the ocular condition.

Ocular examination demonstrated bilateral blindness. The eyes rotated fully in all directions, but incoordinate, irregular, purposeless movements were seen, increasing on lateral gaze. The pupils were fixed, each being 4 mm. in diameter. The media were clear; the disks (fig. 5) were vertically oval and pale. Their size was about one-third that of a normal disk. They were five times the diameter of the central artery, and the fovea was 5 disk diameters away. The disks were well defined temporally, with a pigment ring and scattered pigment extending a short distance temporally. The nasal margins were slightly distorted, giving somewhat the appearance of a pulling of the retina onto the disks. Nasal and adjacent to the disks the retina was considerably lighter in color. The central

vessels were large in comparison to the disks. A large opticociliary vein was seen disappearing at the temporal margin of the right disk. The peripheral branches were normal. The fundal reflexes were normally pink. The maculas were granular in appearance, with the foveas unusually dark. No lesions of the fundus were seen.

Studies included taking of roentgenograms of the skull and optic canals, which were normal. The Wassermann reaction of the blood was negative. Blood counts and urinalyses revealed no abnormality.

COMMENT

In reviewing the literature on aplasia of the optic nerve considerable confusion was seen to exist. Study of the embryology of the optic nerve indicates that aplasia could come from either of two sources, because the nerve arises from mesoderm and from neural ectoderm. The paraxial mesoderm first grows into the fetal fissure, laying down the hyaloid vessels and connective tissue. The fetal fissure then closes from the rim of the optic cup to the hyaloid vessels, the site of the future optic papilla. If the mesoderm failed to enter the fissure, complete closure might occur, preventing subsequent growth of nerve fibers into the optic stalk after they developed from the ganglion cell layer of the retina. This would result in true aplasia, or complete absence, of the optic nerve, not even the central vessels being present. Evidence supporting this has been cited.

If development has proceeded normally except for failure of development of the ganglion cell layer of the retina, the optic nerve head, though small because of absence of nerve fibers, should be present, as evidenced by the central vessels. Such an optic nerve head is seen clinically in well developed eyes, and in the case reported here the condition was bilateral. Brière,[6] Schwarz [14] and Ridley [15] have also reported cases of the bilateral condition. Cords [1] and Velhagen [13] have reported cases in which it was unilateral. Schwarz's patient most closely resembled ophthalmoscopically the patient reported on in this paper, but in his patient only the uncrossed optic fibers were absent.

The condition is observed in patients with poor vision or blindness since birth who show small pale disks, the central vessels being present. The disks may be cupped. Atrophy of the optic nerve must be excluded; a history of normal development and the absence of systemic and neurologic disease are helpful diagnostic points in ruling it out.

SUMMARY

The nature of the embryologic disturbance of partial or complete aplasia of the optic nerve is discussed.

Five cases of partial aplasia, or hypoplasia, of the optic nerve in otherwise normal eyes were collected from the literature, and 1 case of the bilateral condition is reported.

The condition must be differentiated from atrophy of the optic nerve.

TWO NEW VISUAL TEST CHARTS

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Examinations for spectacles involve much more than ophthalmometric and retinoscopic measurements. The refractionist who relies too strictly on such measurements will make many mistakes. The ophthalmometer measures the paracentral and not the central area of the cornea. Away from the center the corneal curvature often tends to become parabolic or ellipsoid rather than spherical. Retinoscopy also suffers from inaccuracies. Refraction may vary through different parts of the pupil. The nonmacular portions of the retina are more readily illuminated than the macula. The area of illumination usually is as large as the macula and therefore much larger than the fovea, which is a depression within the macula and which may differ somewhat from the macula in refraction. This would explain, among other things, why some eyes require appreciably more minus spherical power than is indicated by the results of skiaskopic examination.

Objective measurements being subject to error, considerable reliance must be placed on subjective findings at the trial case. A variety of test charts is required. Each examiner will have his own preferences, and all patients will not respond equally well to the same type of chart. The various test charts available commercially seldom meet all requirements, and the ophthalmologist should supplement them by charts of his own design, drawn with the aid of a ruler, a compass and india ink. Two such charts, which I have found useful, will be described. They involve no new ideas but merely represent what seem to be convenient and reliable arrangements. Before describing them it may be of interest to consider the theoretic foundation for visual test charts.

THEORETIC BASIS FOR VISUAL TEST CHARTS

The most important element in human vision is the recognition of form, or contour. When light acts on the retina sensations are produced which can be grouped into three categories. Arranged in the probable order of phylogenic priority, they are: the light sense, the form sense and the color sense. The light sense deals with the perception of light as such and of variations in the intensity of light. The form sense deals with the recognition of the shapes of things outside the eye. It results in part from the ability of the individual rods and cones to distinguish between stimulation and absence of stimulation and

to this extent may be regarded as a refinement of the light sense. However, the form sense is only partly a retinal function. It depends, among other things, on the dioptric apparatus of the eye for clear images and on the cerebral cortex for visual judgments.

Most diseases of the eye and of the visual nerve paths impair the form sense. Errors of refraction do likewise. Therefore, it is desirable to have an easy way of appraising the form sense at its lower, or physiologic, level. This is readily done by measuring the visual acuity, or determining the smallest retinal images whose contours can be discriminated.

Visual acuity is largely, though not entirely, dependent on the resolving power of the macula. In an optical instrument, such as a telescope, "resolving power" refers to the least possible angular separation of two point sources of light (two stars, for example) that will allow separate images, any lesser separation producing a single image. Likewise, for the eye resolving power is measured by the smallest angular distance, subtended at the nodal point of the eye, at which two points still can be recognized as being separate. This is known as the "minimum separable angle."

At the macula, where the cones are crowded together as closely as possible, each cone is believed to be connected with a single ganglion cell and thus with one axon or fiber of the optic nerve. Theoretically, if two separate points are to be discriminated by the retina their images must be separated at least by the diameter of one macular cone. One intervening cone must remain unstimulated while the cone on either side is excited. This means, disregarding the phenomenon of diffraction, that the diameter of a retinal cone determines the minimum visual angle. Histologic measurements of the average diameter of a macular cone have varied from 0.004 mm. in earlier times to 0.002 mm. in more recent times. By simple trigonometric calculations, with use of Gullstrand's values for the dioptric system of the eye as applied to the reduced eye, these distances are found to subtend angles of 50 and 25 seconds respectively at the nodal point of the eye. Measurements of the minimum separable have given angles as low as 50 seconds for points and 37 seconds for lines.

The "minimum separable angle," which is determined experimentally, and its theoretic counterpart, the "minimum visual angle," which is obtained mathematically, refer to only one type of retinal resolving power —the recognition of the separateness of points or lines. There are other kinds of resolving power, for example, the "minimum distinguishable change of contour," which refers to the recognition of a break in a straight contour when the new contour is parallel and lateral to the original. This faculty of the retina is believed to depend, first, on the complete covering and stimulation of several cones lying in a straight

line by the image of the straight edge of the contour, and secondly, on the sudden partial (but sufficient for purposes of stimulation) covering of the next row of parallel cones by the advanced or recessed edge of the contour or line. Thus, because an image need cover only part of a cone in order to stimulate it, small abrupt increases or decreases in the widths of contours that subtend images smaller in breadth than the diameter of a retinal cone can be discerned. Experimentally, sudden increases in thickness or widths of contours can be recognized which subtend angles as small as 8 or 10 seconds at the nodal point of the eye. An angle of 10 seconds corresponds to a retinal image of 0.0007 mm. This faculty of the form sense, called the "minimum distinguishable change of contour," is used for alining verniers and range finders. Clinically it is not measured, since its variation in acuity parallels that of the minimum separable, and tests of visual acuity based on the latter are much more easily and speedily performed.

Snellen, the originator of modern visual test types, based them on a minimum visual angle of 1 degree. Although it now is known that the minimum visual angle is smaller than this, the 1 degree angle has been retained because it is satisfactory in actual practice and too well established to change. Snellen's test types are capital letters in roman script, so constructed that each letter can be circumscribed by a square which subtends a 5 minute angle at the nodal point of the eye. Each line in a letter and each space between two lines must be of such breadth that a 1 minute angle is subtended at the eye. At the same time, all breaks between lines should, as far as possible, also subtend a 1 minute angle. Thus, in order to discriminate the constituent parts of a letter the eye must be able to resolve them down to a minimum separable of 1 degree.

Letters as test types have the disadvantage of being complicated. They bring in psychologic factors. Letters that are indistinctly seen often can be guessed from their general configuration. Circles containing breaks that subtend a 1 minute angle are more accurate and exclude guessing. However, they are inconvenient in actual use, because they require too much time for identification of the breaks. Preliminary explanation to the patient and, probably, frequent trips to the chart by the examiner are required in order to identify the positions where the patient believes the breaks to be located. This is slow and exasperating and is unsuited to practical clinical work. Capital letters make visual testing easy and speedy and therefore always will be preferred to broken circles or similar test, outlines, such as the capital letters E and U in various positions.

ASTIGMATIC FANS AND DIALS

Some patients with astigmatism find it difficult to determine the correct position of a cylinder by observing either the clarity of a letter

or the absence of tilt. For such patients an astigmatic fan may be easier to use. Other astigmatic patients, at the trial case, will focus on, or if under the influence of a cycloplegic will concentrate on, either the plane of least diffusion or one of the focal planes. In this way, if the astigmatism is not large, letters may be identified to a fair degree and the examiner may be misled into assuming that no astigmatism is present and may diagnose only low normal visual acuity—say 20/25 or 20/30 +. In these circumstances also an astigmatic fan probably would lead to greater accuracy.

A line appears sharply defined when it is properly refracted by the meridians at right angles to it. If a horizontal line is to appear distinct it must be properly focused by the vertical meridians. Because the refracting meridian of a cylindric lens is at right angles to the axis, it may be said that if a line in a chart designed for use in cases of astigmatism appears more poorly defined or grayer than any of the other lines, the direction of this line indicates the approximate axis of the correcting lens. Conversely and more conveniently (because it is easier to select the blackest and most sharply defined line than to select the least black and most blurred) the direction of this line indicates the approximate axis of the correcting lens. Conversely, and more conveniently because it is easier to select the blackest and most sharply defined line than to select the least black and most blurred, the darkest and most sharply defined line is at right angles to the axis of the required cylinder. The examiner should remember to project an axis on the chart to the corresponding axis of the patient's eye. For example, if the darkest line on an astigmatic fan is in the 60 degree axis, this corresponds to the 120 degree axis in the patient's eye, and the correcting cylinder must be placed at 30 degrees in the trial frame.

In an overcorrected myopic eye (artifically hypermetropic), in an overcorrected hypermetropic eye (thereby made myopic) or in an excessively accommodating hypermetropic eye (myopic as a result) the darkest and most distinct lines correspond to the axes of the necessary correcting cylinders that are required after the overcorrection has been abolished. The reason for this becomes apparent when it is recalled that in the presence of astigmatism there are two focal planes. If by overcorrection with spheres or by excessive accommodation these planes both are shifted from one side of the retina to the other, the focal plane which initially was nearer to the retina and therefore less ametropic becomes the one that is farther from the retina and more ametropic. Spherical lenses merely move the two focal planes simultaneously either forward or backward and retain the same distance between them. Only a cylindric lens can bring one plane closer to the other.

Astigmatic fans do not give accurate results when the eye is in a condition of mixed astigmatism. By concentrating or focusing on the plane

of least diffusion in the interval between the two focal planes the patient will see all lines equally sharply, though he will see the chart as a whole blurred to a variable degree. In the presence of mixed astigmatism it is better to fog the patient's vision with plus spheres and then to use minus cylinders; or, since mixed astigmatism is preeminently the condition for the crossed cylinder, it may be better to use the astigmatism chart only for position.

Before an astigmatic fan is used for myopes, slightly less (by 0.25 to 0.5 D.) than the approximate required spherical correction as discovered by previous retinoscopic study should be placed in the trial frame. Patients with absolute hypermetropia (hypermetropia that cannot be overcome by accommodation) also should have their vision slightly undercorrected for the absolute portion of the hypermetropia. Those with facultative hypermetropia (hypermetropia that can be overcome by accommodation) should have the vision blurred with plus spheres and then corrected with minus cylinders.

DESCRIPTION OF SUGGESTED CHARTS

The charts are depicted in the accompanying photographs. Chart 2 is merely a supplement to the large astigmatic fan dial of chart. 1.

In chart 1 all letters ordinarily curved or partly curved, such as C, B, D, G, O, P and U, were drawn with all lines straight and in the horizontal and vertical directions. This was done to eliminate any aid to identification which curves may furnish. These letters when added to those in which there are only horizontal and vertical lines, namely, E, F and H, provide sufficient variety. Letters with oblique lines, such as N, V, X, Y and Z, were included because often they are helpful in establishing the axes in cases of astigmatism. When the axes are incorrect they are apt to appear distorted.

Letters were drawn to the correct size [1] for distances of 200, 100, 70, 50, 40, 30, 25, 20, 15 and 10 feet (60, 30, 21, 15, 12, 9, 7.5, 6, 4.5 and 3 meters). A second line each of the size used at 20 and at 15 feet was placed at the bottom of the card to help prevent memorization.

1. One minute angles are subtended at the following distances by lines drawn to the indicated widths:

At	200	feet—by	widths of	18.3	mm.
At	100	feet—by	widths of	9.1	mm.
At	70	feet—by	widths of	6.4	mm.
At	50	feet—by	widths of	4.6	mm.
At	40	feet—by	widths of	3.7	mm.
At	30	feet—by	widths of	2.7	mm.
At	25	feet—by	widths of	2.3	mm.
At	20	feet—by	widths of	1.8	mm.
At	15	feet—by	widths of	1.4	mm.
At	10	feet—by	widths of	0.9	mm.

At the ends of the lines for 50, 40, 30 and 25 feet there are broken circles with double breaks, drawn to the same scale. They are helpful in establishing the axis in cases of astigmatism, the correct position being the one where both breaks are most clearly defined.

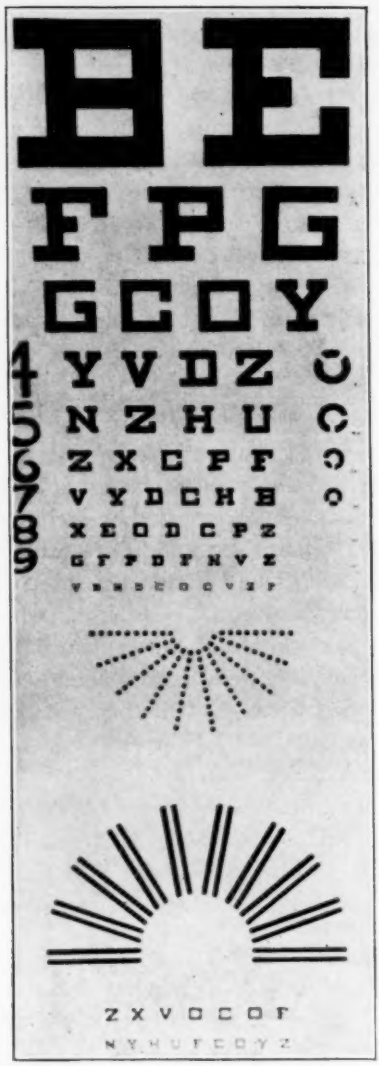

Chart 1.—Test chart with rows of letters arranged for testing at varying distances. Two astigmatic fan dials are included.

Two astigmatic fan dials are included. The larger, and more useful, one was drawn so that the width of each line subtends a 1 minute angle at 40 feet. It is composed of double lines separated by spaces of the

same width as the lines. Double line fans are easier to discriminate than single line configurations. When a double line is blurred the intervening space appears smudgy, and this provides a factor in addition to the blackness or the sharpness of lines by which the patient may judge. Only nine meridians are represented at intervals of 20 degrees. More meridians would tend to obscure judgment by providing too many lines for observation and comparison.

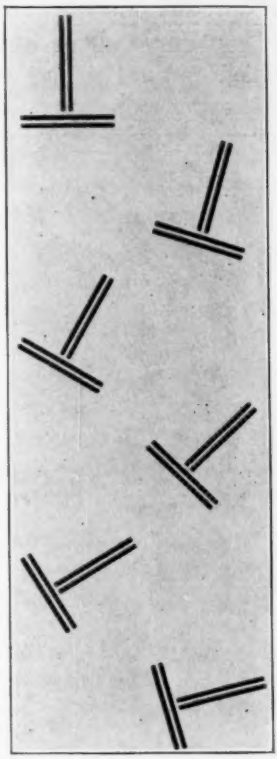

Chart 2.—Test chart supplementing the large astigmatic fan dial in chart 1. See text for further explanation.

The smaller, and less useful, astigmatic dial was drawn to subtend a 1 minute angle at 20 feet. It is useful sometimes for detecting and correcting small astigmatic errors. Each line consists of a series of breaks and short lines, all of the same length and all subtending both in length and in width a 1 minute angle at 20 feet. Any line that is not in focus will appear smudgy, because if the broken line components are not properly focused by the meridians at right angles to the line they will blur the clear spaces of the breaks. Thus, the interpretation of this

dial is just the opposite from that of the large double line fan. For nonaccommodating, spherically undercorrected eyes the most smudged or most continuous-appearing line requires a correcting cylinder with its axis at right angles to the direction of the smudged line.

Chart 2 contains six figures, each consisting of two pairs of lines at right angles to each other. The lines subtend a 1 minute angle at 40 feet. After the patient has selected the pair of lines that is darkest and most clearly defined he is asked to look only at that particular figure. Then, while cylinders are inserted into the trial frame, he decides when the two pairs of lines are equal in blackness and in definition.

Astigmatic dials that subtend a 1 minute angle at 40 feet are not suitable for the larger astigmatic errors, over 1.5 D. For these it would be desirable to have similar dials drawn to a larger size.

OSTEITIS DEFORMANS WITH ANGIOID STREAKS

REPORT OF A CASE

W. HOWARD MORRISON, M.D.
OMAHA

Paget [1] in 1877 reported on a form of chronic inflammation of the bones. He ascribed the name osteitis deformans to this disease. The patient described by Paget, in addition to having skeletal changes, had become somewhat deaf, and the "sight was partially destroyed by retinal hemorrhage first in one eye then in the other."

Osler [2] found Paget's disease of the bone in 2 of 20,000 patients examined at the Johns Hopkins Hospital. Schmorl [3] discovered definite microscopic evidence of osteitis deformans in 3 per cent of 4,614 autopsies performed on persons over 39 years of age. Paget's disease of the bone is therefore rarely seen clinically but appears frequently as a subclinical entity.

The characteristic eyeground appearance of what is now known as angioid streaks was first described and illustrated by Doyne [4] in 1889. Two years later Plange [5] described a case. Knapp [6] in 1892 cited Plange's publication and reported a similar case of retinal striae. Knapp termed the retinal changes angioid streaks. In 1938 Goedbloed [7] found that since 1889 "about 140 cases of this condition have been reported in the literature."

Verhoeff [8] stated that the pathologic picture observed by him of angioid streaks consisted of "ridges comprising the inner layers of the

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1. Paget, J.: On a Form of Chronic Inflammation of Bones (Osteitis Deformans), Med.-Chir. Tr., London **60**:37-63, 1877.

2. Osler, W.: Principles and Practice of Medicine, revised by T. McCrae, ed. 11, New York, D. Appleton and Company, 1930, pp. 1169-1170.

3. Schmorl, G., cited by Terry, T. L.: Angioid Streaks and Osteitis Deformans, Tr. Am. Ophth. Soc. **32**:555-573, 1934.

4. Doyne, R. W.: Choroidal and Retinal Changes the Result of Blows on the Eyes, Tr. Ophth. Soc. U. Kingdom **9**:128, 1889.

5. Plange, O.: On Pigment Striae with Secondary Changes in the Retina After Hemorrhage, Arch. Ophth. **21**:282-288, 1892.

6. Knapp, H.: On the Formation of Dark Angioid Streaks as an Unusual Metamorphosis of Retinal Hemorrhage, Arch. Ophth. **21**:289-292, 1892.

7. Goedbloed, J.: Syndrome of Groenblad and Strandberg, Arch. Ophth. **19**:1-8 (Jan.) 1938.

8. Verhoeff, F. H.: The Nature and Pathogenesis of Angioid Streaks in the Ocular Fundus, Tr. Sect. Ophth., A. M. A., 1928, pp. 243-263.

choroid, produced by cicatricial contraction of fibrous tissue which had replaced the deeper layers. The fibrosis involved almost uniformly about one half the total extent of the choroid and was associated with extensive obliteration of the vessels and a few subchoroidal hemorrhagic extravasations."

The eye examined by Verhoeff was not definitely accepted by Troncoso [9] as showing angioid degeneration. An opaque cornea precluded ophthalmoscopic examination. The retinal streaks were seen microscopically only after the eye had been enucleated.

Benedict [10] in 1936 enucleated the blind glaucomatous right eye of a patient who had typical angioid streaks in the left eye. Ophthalmoscopic examination of the blind eye could not be done because of the cloudy cornea. The enucleated eye was fixed and divided at the equator. Angioid streaks were then seen by means of a Zeiss binocular magnifier and a Shahan lamp. The posterior half of the eye was embedded in colloidin (a pyroxylin preparation), sectioned in the equatorial plane and examined microscopically. No pathologic changes were observed which could be identified as angioid streaks.

Law [11] sectioned and examined an eye in which he had seen angioid streaks. He concluded "that angioid streaks are in some cases at least due to a plication of the retina, separation occurring between the layer of rods and cones and the pigment epithelium, the intervening space being filled by pigmentary debris." Law added: "There is no necessity to invoke the occurrence of clefts in Bruch's membrane in explanation of angioid streaks since none is present in this case."

Hagedoorn [12] observed angioid streaks in the eye of a patient with pseudoxanthoma elasticum. The patient soon died; the eyes were enucleated, and a histologic study was made. Degeneration of Bruch's layer with ruptures in this degenerated membrane was observed. Hagedoorn concluded that angioid streaks are ruptures in a degenerated Bruch's membrane. Verhoeff,[13] however, took exception to this conclusion, because he (Verhoeff) and Sisson "had reported and depicted changes in Bruch's membrane essentially identical with those found

9. Troncoso, M. U.: Internal Diseases of the Eye and Atlas of Ophthalmology, Philadelphia, F. A. Davis Company, 1939, p. 369.

10. Benedict, W. L.: The Pathology of Angioid Streaks in the Fundus Oculi, J. A. M. A. **109**:473-475 (Aug. 14) 1937.

11. Law, F. W.: Diseases of the Retina and Optic Nerve—A Contribution to the Pathology of Angioid Streaks, Tr. Ophth. Soc. U. Kingdom **58**:191-207, 1938.

12. Hagedoorn, A.: Angioid Streaks, Arch. Ophth. **21**:746-774 (May); 935-965 (June) 1939.

13. Verhoeff, F. H.: Angioid Streaks, Correspondence, Arch. Ophth. **22**:313 (Aug.) 1939.

by Hagedoorn." They concluded that "the condition is without pathologic significance and is simply a senile change which usually does not impair the function of the eye." Verhoeff also questioned Hagedoorn's ophthalmoscopic diagnosis of angioid streaks.

Groenblad [14] published in 1929 a report on 3 patients with angioid streaks and pseudoxanthoma elasticum. This report was presented as proof that angioid streaks are merely manifestations of a general disease of the elastic substance.

Goedbloed [7] in 1938 gathered from the literature 57 cases in which angioid streaks in the fundus accompanied typical pseudoxanthomatous involvement of the skin. In 2 cases they accompanied senile elastosis, a degeneration of both elastic and collagenous connective tissue of the dermis.

The occurrence of angioid streaks in 9 cases of Paget's disease was reported in 1934 by Terry.[15] He studied 22 patients with osteitis deformans and found 4 to have angioid streaks. The other 5 cases of Paget's disease with angioid streaks were collected from the literature. Among these was the case alluded to by Verhoeff [8] in 1928. Lambert [16] in 1939 reported 2 additional cases.

The 11 cases of angioid streaks associated with Paget's disease are reported as follows:

Verhoeff [8]	1928
Rowland (cited by Terry [15])	1929
Juler (cited by Batten [17])	1931
Berliner (cited by Terry [15])	1933
Vail (cited by Terry [15])	1934
Terry [15] (4 cases)	1934
Lambert [16] (2 cases)	1939

Pigmented spots, streaks, arteriolar sclerosis, retinal and choroidal hemorrhages and cataracts have been seen in the eyes of patients with osteitis deformans. Peppmüller [18] and von der Heydt [19] have each

14. Groenblad, E.: Angioid Streaks—Pseudoxanthoma elasticum; vorläufige Mitteilung, Acta ophth. **7**:329, 1929.

15. Terry, T. L.: Angioid Streaks and Osteitis Deformans, Tr. Am. Ophth. Soc. **32**:555-573, 1934.

16. Lambert, R. K.: Paget's Disease with Angioid Streaks of the Retina, Arch. Ophth. **22**:106-109 (July) 1939.

17. Batten, R. D.: Angioid Streaks, and Their Relation to a Form of Central Choroidal Disease, Brit. J. Ophth. **15**:279-289, 1931.

18. Peppmüller, F.: Ueber eine bisher nicht beschriebene beiderseits symmetrisch gelegene subepitheliale Hornhauttrübung bei einem Fall von Pagetscher Ostitis deformans, Klin. Monatsbl. f. Augenh. **98**:1-14, 1937.

19. von der Heydt, R.: Osteitis Deformans with Corneal Degeneration, Am. J. Ophth. **20**:1139-1141, 1937.

reported pigmented corneal degeneration in patients suffering from Paget's disease of the bone.

Except for the level of serum phosphatase, which is increased in the presence of osteitis deformans [20] beyond a normal 4 Bodansky units, laboratory data are of questionable value. Jaffe [21] stated that the microscope will show definite osteitis deformans before the diagnosis can be made by roentgen examination.

I could find no report of angioid streaks, pseudoxanthoma elasticum and Paget's disease of the bone occurring in the same patient.

REPORT OF CASE

P. E., a 64 year old retired packing house executive, was first seen by me in October 1940. He came in for a refraction simply because his reading glasses had broken. He was born in Council Bluffs, Iowa, of German parents. There were five boys and four girls, all healthy, in the family. The patient had one child, a son, who was normal. There was no familial history of Paget's disease as far back as the patient could trace.

At 35 years of age P. E. began having pains in the leg, of unknown origin. When he was 40 it was noted that he required a larger hat than usual and that hearing was no longer present in the left ear. No head noise was noted at this time. Five years later the patient began to hear a locust-like sound in his ears. At this time his hearing on the right began to fail noticeably. Complete deafness occurred in 1932. The head noises ceased except for a few bars of music heard occasionally, which seemed to be repeated over and over again. Pain low in the back was experienced in 1928. Shortly thereafter a ureteral calculus was passed. The pain in the lower part of the back continued, and a roentgenogram was taken, which revealed new bone formation in the sacrum. A diagnosis of Paget's disease, however, was not made until the patient entered the Veterans' Hospital at Lincoln, Neb., in 1930.

The patient at one time had been 5 feet 6 inches (168 cm.) tall. On examination he measured 5 feet 3 inches (160 cm.). His weight, 160 pounds (72.6 Kg.), had not changed during the past twenty-five years. At one time he had worn a size 6⅞ hat; at the time of examination he required something over a size 8. The color of his skin had remained the same. He had never had any visual difficulty except the need of glasses for near work.

The patient presented a typical picture of osteitis deformans. His head was very large and his legs markedly bowed. Roentgen examination by Drs. Tyler and Simonds revealed increased density, with exaggeration of the linear markings of the sacrum, both innominate bones and both femurs. The right acetabulum and the neck of the right femur were deformed. The skull showed great thickening of the outer table, with increase of the anteroposterior diameter of the head and thickening of the floor of the skull. The sella turcica was deformed.

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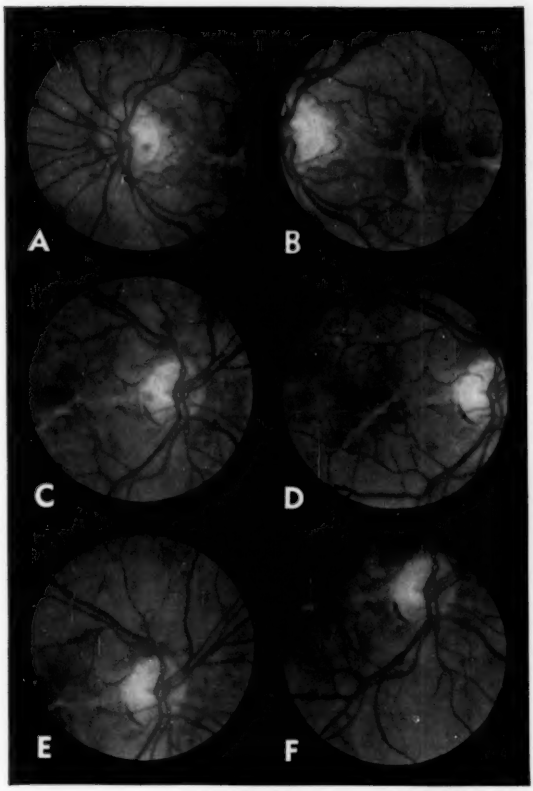
20. Gutman, A. B.; Tyson, T. L., and Gutman, E. B.: Serum Calcium, Inorganic Phosphorus and Phosphatase Activity in Hyperparathyroidism, Paget's Disease, Multiple Myeloma and Neoplastic Disease of Bones, Arch. Int. Med. **57**:379-413 (Feb.) 1936.

21. Jaffe, H. L.: Paget's Disease of the Bone, Arch. Path. **15**:83-131 (Jan.) 1933.



Angioid streaks in a case of osteitis deformans. See text for explanation.
(The illustrations were reversed in reproduction.)

UNIV. OF MICH.

The phosphatase content of the blood serum was normal (3.8 Bodansky units). Dr. O. J. Cameron, a dermatologist, could find no evidence of pseudoxanthoma elasticum.

The ocular findings were as follows:

The vision in each eye without correction was 20/20. Jaeger test type 1 was easily read with the addition of + 2.00 D. spheres. A very moderate degree of compound hyperopia was present in each eye.

Externally the eyes were not remarkable. The pupils were round, regular, equal and active to light and in accommodation.

Corneal microscopic study demonstrated (except for arcus senilis) perfectly normal corneas. The irises were somewhat atrophic, and the lenses were clear except for a slight amount of gray nuclear sclerosis.

The peripheral fields of vision disclosed superior bitemporal defects. These defects were from 20 to 30 degrees wide and were midway between the point of fixation and the periphery.

The central fields of vision at 2 meters with a 1 mm. white target failed to disclose any scotoma about the point of fixation. The blindspots were somewhat enlarged.

Ophthalmoscopic Examination.—Right Eye: The media were clear; the disk was round and flat; the temporal half of the papilla was somewhat pale, and the nasal margin was blurred. Spontaneous venous pulsation was visible. Slight arteriovenous compression was noted. The arteries were somewhat irregular in caliber (*A* in accompanying illustration).

The disk was surrounded by a yellow-gray band ("halo"). This ring of retinal disturbance was particularly well seen just temporal to the papilla. A very small red line resembling a capillary came off or began just under the disk portion of the inferior temporal vein. This red line coursed upward and slightly temporally and then returned to the papillary border at 3 o'clock. It then proceeded in a slightly inferior and temporal direction for about 1 disk diameter. The red line in its course varied in width from ⅛ to ½ vein diameter and had on each side, after it left the papilla, a broad gray-yellow border.

One disk diameter temporal to the papilla the yellow streaklike retinal defect divided into three parts (*B*). One streak had a directly superior extension ending in a group of small, round gray areas of retinal disturbance. Another yellowish defect coursed inferiorly and temporally, and another had a directly temporal course. The point of division of these yellow retinal streaks was approximately in the center of the macular area.

There extended superiorly from the disk at 11 o'clock a broad red line which varied in caliber from ½ to 1 vein diameter.

Another very fine red streak, also deep to the retinal vessels, left the disk at 4 o'clock, proceeded nasally and inferiorly, broadened into a gray-yellow band and ended about 3 disk diameters away in a clump of pigment.

Left Eye: The media were clear; the disk was flat and somewhat oval, with a nasal marginal blur. A narrow strip of choroidal pigment bordered the disk from 7 to 9 o'clock (*C*). Just off the disk at 5 and 2 o'clock were solitary small clumps of black pigment. Proceeding from an incomplete yellow temporal marginal disk ring, there coursed from the papilla at 1 o'clock a rather broad gray streak, deep to the retinal vessels. This streak proceeded temporally for 1 disk diameter (*D*); then it dipped somewhat inferiorly, just missed the macula and gradually disappeared. Proceeding from the disk, at about 12 o'clock (*E*), another, redder retinal streak, progressed superiorly and nasally about 2 disk

diameters. Another irregular red streak (*F*), bordered on each side by a gray-yellow band, left the disk at about 5 o'clock and proceeded inferiorly and nasally for about 4 disk diameters.

COMMENT

A twelfth case of osteitis deformans with angioid streaks is reported; the description is the first to be accompanied with colored photographs of the fundus.

The retinal involvement was typical, extensive and bilateral. The macula of each eye was just spared. Cutaneous lesions typical of pseudoxanthoma elasticum were not present.

Subclinical osteitis deformans occurs frequently. Patients with angioid streaks should therefore be examined not only for pseudoxanthoma elasticum but for evidence of Paget's disease. The normal phosphatase content of the serum of the patient in this case probably indicates that the disease is quiescent.

The pathology of angioid streaks has not as yet been definitely determined.

Serial color photographic studies of this patient's fundi are contemplated.

Dr. William H. Stokes took the photographs which accompany this article.

BLINDNESS IN THE STATE OF NEW YORK

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Statistics on the causes and on the prevalence of blindness in the United States are so few that it is doubtful whether we have anything near an appropriate conception of either. On the causes of blindness there are only the reports which have come from Missouri, Colorado, Illinois, New Jersey, Pennsylvania, Washington, Louisiana and Indiana in the past twenty years and one report on the causes of blindness in children. These are lacking in uniformity and cover relatively small groups of cases, of which only small percentages are included in the etiologic tables. On the prevalence of blindness the federal census report cannot be considered at best more than a rough and misleading estimate, owing partly to the absence of ophthalmic examinations. Approximate figures on incidence, based on the annual census of new students entering the various state schools for the blind, are offered by the National Society for the Prevention of Blindness and by the American Foundation for the Blind.

The Social Security Board, through its Bureau of Public Assistance, is concerned with assistance to the blind and with the prevention of blindness and because of its national scope should in time produce statistics which will be more uniform and accurate. It is to be hoped that it will exercise its prerogative to bring about standard practices relating to blindness, so that a fairly accurate conception of the causes and prevalence of blindness in the United States may soon be obtained. But it may not be until blindness is declared a reportable disease that this will be brought about.

The public welfare law of the state of New York sets forth a definition of blindness as follows: "A person shall be considered as blind who has not more than twenty/two hundred visual acuity in the better eye and for whom a diagnosis and medical findings show that vision cannot be improved to better than twenty/two hundred; or where there is loss of vision due wholly or in part to impairment of field vision or

From the New York State Commission for the Blind.

to other factors which affect the usefulness of vision to a like degree. Classification of the eye condition within the provisions of this definition shall be determined according to standards adopted by the state department in consultation with the New York state commission for the blind." This definition is too broad and should be rewritten to give consideration to reading vision, but it corresponds well with that adopted by the Social Security Board. A fault lies in its failure to state the qualifications of those who are to be entrusted with the reporting of blindness. This omission necessarily compromises the value of the data which follow.

The present study of causes of blindness in cases coming under "blind assistance" was undertaken by the Bureau of Services for the Blind of the New York State Department of Social Welfare. This bureau represents the functions and duties of the New York State Commission for the Blind and is charged with the classification of blindness under the legal definition set forth in the foregoing paragraph. A two year period, from July 1937, when the Social Security Act became effective, to July 1939, was covered, and 2,685 cases were analyzed. Much difficulty attended the work because of the incompleteness of many reports and their failure to convey the precise nature of the ocular condition of the blind person described. Much information was acquired through correspondence but in many instances the condition had to be inferred from the meager information of the report. This report is therefore presented to the members of the medical profession with a plea for better reporting of the visual condition of the blind and of the near blind. Accurate data covering a large portion of the population are essential to the establishment of a scientific basis for programs for the prevention of blindness, and the responsibility for acquiring such data rests squarely with the ophthalmologist. Workers studying reports of blindness are constantly hindered by the fact that many physicians believe that the treatment and even the examination of a patient are quite unrelated to the recording of his case. Records are important working instruments—important for the physician, the social worker and the vocational worker in their service to the blind. They are thus important to the blind person himself, in whose interest most of the effort is made. Full, accurate records of ocular conditions in the blind and the near blind are a distinct responsibility of the physician to society and to science, for progress in preventive, curative and restorative ophthalmology cannot be estimated without statistics. It is generally believed, for example, that the incidence of blindness is steadily decreasing in the United States; yet it is impossible to demonstrate this statistically.

Jackson[1] stated that in cases of blindness "a discussion on the extent or the cause of blindness may be the only professional service we can render. Every attempt to assist, to train, to compensate, or to make economic provision for the blind renders the diagnosis of blindness more important . . . To decline to reach and state a definite conclusion as to the existence, degree, and etiology of blindness is, in most cases, equivalent to a confession of incompetence, laziness, or indifference to the question presented. It is a shirking of responsibility for anyone holding himself before the community as an expert as to diseases and defects of the eye and vision, to refuse to give a diagnosis as to blindness, or to state it in such doubtful terms as to render that diagnosis worthless."

The primary purpose of reports of blindness is to qualify the patient or applicant for assistance and to make the various services of the Commission for the Blind available to him. If the reports are adequate and give a clear conception of the patient's condition, that is, his best visual acuity and the condition which is responsible for his visual deficiency, relief is not delayed and all statistical requirements are satisfied. If a clear understanding is not given by the report, eligibility is not attained and aid is necessarily delayed, which causes unnecessary hardship to the patient. The entire program of assistance to the blind in general and to the individual blind person depends on the ophthalmologist.

It should be agreed, however, that the determination of the cause of blindness is occasionally difficult, if not impossible. A patient may have atrophic or disorganized eyeballs or empty sockets and may not be able to give an enlightening history, or the media may be clouded and may interfere with the examination to such a degree that the real cause of blindness cannot be determined. Again, there are many ocular diseases for which the most painstaking investigations fail to reveal an unequivocal cause. Nevertheless, if progress is to be made there must be unremitting effort in every case to establish a diagnosis. Every diagnosis given on a report should fulfil the topographic and etiologic requirements, as, e. g., "chronic tuberculous uveitis" or "diabetic retinitis." The naming of a general disease alone, such as "meningitis" or "syphilis," is, of course, inadequate, since it fails to specify the ocular condition. Likewise, such terms as "keratitis" and "retinitis" are not adequate, for the conditions mentioned, have many forms and a variety of causes. The term "leukoma," though a sufficient explanation for

1. Jackson, E.: The Diagnosis of Blindness, Tr. Sect. Ophth., A. M. A., 1925, p. 319.

poor vision or blindness, is not a suitable diagnosis, for leukoma may result from many different diseases. "Blind" or "congenitally blind" is not a diagnosis. In reporting the effect of physical or chemical trauma on the eye the causative agent should be named and the accident specified as occupational or nonoccupational. Of the latter, it should be stated whether the accident occurred in the home and was thus a "household accident," in traffic and was thus a "traffic accident" or at play and was thus a "play accident." This information on the nature of the trauma is completely lacking in most of the available statistics.

It is the policy of the Commission for the Blind of the state of New York to consider all reports confidential. Reports received at present are carefully checked over for rating of the visual acuity and the visual fields when indicated (with sizes of test objects noted) and for topographic and etiologic diagnosis, are given a rating of "blind" or "not blind," and are coded according to the Standard Classified Nomenclature of Disease. If the report is inadequate it may be returned to the examiner for additional information. The examination forms in use at present ask for a minimum of medical information, and information of purely social value, although of great importance, is not asked of the busy physician.

The Commission for the Blind was created by law in 1913, and approximately 33,346 blind or partially blind persons have been registered by it. In the decade from 1930 to 1939 inclusive, 1,132 of 14,603 patients could not be classified definitely, because of insufficient medical information. In another series, of 17,905 consecutive patients, 3,213 could not be classified as blind or not blind, because of incomplete reports.

Under the public welfare law the care and relief of blind persons are declared special matters of state concern and necessary in promoting public health and welfare. The assistance offered by the state includes aid, care, support, service, medical or surgical care, board in a private institution and "such other aid as the condition of blindness may make necessary." To receive assistance the blind person must establish eligibility. Since the Social Security Act went into effect there have been more than 3,554 recipients of such assistance.

The state commission law makes it mandatory to keep a register of the blind, together with the causes of blindness, and to establish measures for the prevention of blindness as well as to promote the general welfare of the blind. Special services include medical social care of the eyes to improve vision or to prevent further loss of sight; home teaching and case work, including Braille and simple hand occupations; parent education and home training of the blind preschool child; industrial training in the home, and the sale of the products of

Combined Etiologic and Topographic Classification of Blindness (According to the Standard Classified Nomenclature of Disease) of Recipients of "Blind Assistance" Registered from July 1937 to July 1939

Diseases Due to Prenatal Influences		
Eyeball		
Hydrophthalmos; infantile glaucoma	15	
Microphthalmos	12	27
Iris		
Aniridia	7	
Congenital cyst	1	8
Choroid		
Coloboma	1	
Albinism	4	5
Crystalline Lens		
Aphakia, congenital; absence of lens	1	
Cataract, congenital	62	
Luxation, congenital	1	64
Vitreous		
Vestigial structures	1	1
Retina		
Amaurosis, congenital	7	
Amblyopia, congenital	4	
Degeneration, macular, congenital (Best); infantile type of atrophy	9	
Retinitis pigmentosa	103	123
Optic Nerve		
Congenital atrophy	8	8
Errors of Refraction		
Congenital myopia	6	6
Ocular Neuromuscular Mechanism		
Congenital nystagmus	11	11
		253
Diseases Due to Infection		
Eyeball		
Panophthalmitis	9	
Phthisis bulbi, infectional	39	
Disorganized globe, postinfectional	8	56
Cornea		
Keratitis, deep	18	
Keratitis, parenchymatous	23	
Leukoma	270	
Staphyloma	6	
Syphilitic keratitis	4	
Tuberculous keratitis	3	324
Sclera		
Scleritis, anterior, due to unknown infection	1	
Staphyloma, postinfectional	2	3
Iris		
Endophthalmitis	15	
Iritis, iridocyclitis (includes plastic)	63	
Occlusio pupillae (pupillary membrane)	4	
Seclusio pupillae	4	
Syphilis	1	
Uveitis; iritis, endogenous, due to remote (focal) infection	21	
Uveitis, sympathetic	2	
Uveitis, syphilitic	5	115
Choroid		
Choroiditis, central	7	
Choroiditis, disseminated	22	
Choroiditis, disseminated, infection unknown	19	
Choroiditis, suppurative	2	
Choroiditis, sympathetic	31	
Choroiditis, tuberculous	2	83
Crystalline Lens		
Cataract due to uveitis, infection	7	7
Vitreous		
Opacities due to remote infection	6	
Opacities due to syphilis	1	7
Retina		
Atrophy, postinfectional	1	
Chorioretinitis, syphilitic	3	
Detachment due to infection of uveal tract	6	
Hemorrhage due to infection	2	
Tuberculous periphlebitis	1	
Retinitis proliferans, infectional	1	
Retinitis, syphilitica; angiopathia syphilitica	3	17
Optic Nerve		
Atrophy, infectious	28	
Atrophy due to syphilis (including tabes dorsalis)	135	
Neuritis	2	
Meningococcic neuritis	2	
Retrobulbar neuritis, infectional	2	
Syphilitic neuritis	1	
Meningococcic atrophy	3	173

Combined Etiologic and Topographic Classification of Blindness (According to the Standard Classified Nomenclature of Disease) of Recipients of "Blind Assistance" Registered from July 1937 to July 1939—Continued

Glaucoma		
Secondary glaucoma, postinfectional	7	7
Conjunctiva		
Conjunctivitis, chronic nonspecific	1	
Conjunctivitis, infectious, acute	5	
Gonococcic conjunctivitis	2	
Trachoma	10	
Trachoma, chronic	10	28
		820
Diseases Due to Intoxication		
Optic Nerve		
Toxic amblyopia	3	
Toxic amblyopia, due to ethyl alcohol	2	
Toxic amblyopia, due to methyl alcohol	3	8
		8
Diseases Due to Trauma or Physical Agents		
Eyeball		
Injury	7	
Penetrating wound	3	
Phthisis bulbi, traumatic	31	
Anophthalmos, postoperative	37	
Perforating wound	1	79
Cornea		
Burn (lime, etc.)	7	
Staphyloma, post-traumatic	2	9
Sclera		
Staphyloma due to chronic glaucoma	1	
Staphyloma due to myopia	4	5
Iris		
Cyclodialysis	2	2
Choroid		
Rupture	1	
Choroidal changes in myopia	46	47
Crystalline Lens		
Dislocation	2	
Aphakia, post-traumatic (real loss of vision not known)	1	
Aphakia, postoperative (real loss of vision not known)	21	
Secondary cataract	8	
Secondary cataract, postoperative	3	
Traumatic cataract	10	45
Retina		
Detachment due to myopia	19	
Detachment, traumatic	6	
Hemorrhage, traumatic	4	
Detachment, postoperative	5	34
Optic Nerve		
Atrophy due to pressure of arteriosclerotic arteries (senile atrophy)	1	
Atrophy, post-traumatic	19	
Block (including atrophy) from pressure of neoplasm	8	
Papilledema due to pressure (real loss of vision unknown)	4	
Atrophy due to pressure	19	
Retrobulbar neuritis due to pressure	2	53
Glaucoma		
Secondary glaucoma, post-traumatic	3	
Secondary glaucoma, postoperative	1	4
		278
Diseases Due to Circulatory Disturbances		
Choroid		
Atrophia gyrata of choroid and retina	1	1
Vitreous		
Hemorrhage	6	6
Retina		
Arteriosclerotic disease	14	
Atrophy due to thrombosis	1	
Detachment due to hemorrhage	3	
Hemorrhage due to blood dyscrasia	1	
Hemorrhage, preretinal, cause unspecified	4	
Retinitis of hypertension	3	
Retinitis nephritica	3	
Retinitis proliferans	3	32
Optic Nerve		
Atrophy due to circulatory disturbance	12	
Papilledema due to increased intracranial pressure, atrophy	12	24
		63

Combined Etiologic and Topographic Classification of Blindness (According to the Standard Classified Nomenclature of Disease) of Recipients of "Blind Assistance" Registered from July 1937 to July 1939—Continued

Diseases Due to Disturbances of Innervation or of Psychic Control		
Ocular Neuromuscular Mechanism		
Nystagmus of amblyopia	9	9
		9
Diseases Due to or Consisting of Static Mechanical Abnormality		
Crystalline Lens		
Luxation, spontaneous	15	15
Retina		
Embolism of retinal artery	2	2
Glaucoma		
Acute primary glaucoma	13	
Chronic noncongestive glaucoma	173	
Chronic congestive glaucoma	3	
Secondary glaucoma due to displacement of lens	2	
Secondary glaucoma due to swelling of lens (in cataract)	20	211
Errors of Refraction		
Astigmatism	3	
Myopia due to increased refraction of nucleus of lens	1	
Myopia, progressive	17	
Myopia, high, with degeneration	126	147
		375
Diseases Due to Disorders of Metabolism, Growth or Nutrition		
Cornea		
Keratomalacia	1	1
Crystalline Lens		
Diabetic cataract	19	
Senile cataract	146	165
Retina		
Diabetic retinitis	19	
Macular senile (including presenile) degeneration	3	
Retinitis circinata	1	23
Optic Nerve		
Postretinal atrophy	1	1
		190
New Growths		
Choroid		
Sarcoma	1	1
Optic Nerve		
Atrophy due to astrocytoma	2	2
		3
Diseases Due to Unknown or Uncertain Causes the Structural Reaction to Which Is Manifest		
Eyeball		
Disorganized globe (not postinfectional)	16	
Atrophy, cause unknown	21	37
Cornea		
Degeneration, corneal, familial	1	
Degeneration, hyaline, of old scars of cornea	3	
Dystrophy, epithelial	2	
Keratitis, band	3	
Keratoconus	7	
Pannus degenerativus	7	23
Choroid		
Arteriosclerosis of choroidal vessels	1	1
Vitreous		
Opacities in vitreous due to noninflammatory cellular infiltration	2	2
Retina		
Atrophy, progressive retinal	8	
Atrophy in infantile amaurotic family idiocy (Tay-Sachs)	2	
Atrophy in juvenile amaurotic family idiocy (Spielmeyer-Stock)	1	
Detachment, cause unknown	54	
Degeneration of macula	14	79
Optic Nerve		
Neuritis, cause unknown	6	
Neuritis (retrobulbar) in disseminated sclerosis	2	
Atrophy, cause unknown	282	
Atrophy due to multiple sclerosis	2	292
Glaucoma		
Absolute glaucoma	111	111
Conjunctiva		
Symblepharon	1	
Pemphigus of conjunctiva	1	2
		547

Combined Etiologic and Topographic Classification of Blindness (According to the Standard Classified Nomenclature of Disease) of Recipients of "Blind Assistance" Registered from July 1937 to July 1939—Continued

Diseases Due to Causes Not Determinable in the Particular Case		
Eyeball		
Phthisis bulbi, cause undetermined	28	
Amaurosis	12	40
Cornea		
Ulcer of cornea, cause undetermined	1	
Scar of cornea, cause undetermined	2	
Keratitis, unknown	3	6
Iris		
Iritis, cause undetermined	3	3
Crystalline Lens		
Cataracta complicata	11	11
Vitreous		
Opacities in vitreous, cause undetermined	8	8
Retina		
Retinal hemorrhage, cause undetermined	1	
Retinitis, cause undetermined	12	
Chorioretinitis, cause undetermined	55	68
Glaucoma		
Secondary glaucoma, cause undetermined	3	3
		139
Grand Total		2,685

blind workers throughout the state. Over 2,000 Federal talking book machines are distributed through the commission. Acute ophthalmia is a special interest of the commission. Of 278 cases reported by the state department of health between July 1, 1938 and June 30, 1939, blindness resulted in only 1.

In this study, which covers a two year period, many sources of error were encountered in addition to those already indicated. After due study it was decided to report on the cause of blindness for each person rather than for the total number of blind eyes. There is a vast difference between one blind eye and two blind eyes. This basis for reporting was thought better, too, because a high percentage of reports listed one eye as "enucleated" without describing its condition at the time of enucleation. The cause of blindness was considered to be that which produced blindness in the second, or remaining, eye. This policy necessarily introduced a minor source of error, for in the small percentage of cases in which the causes were different for the two eyes it could not always be determined accurately which was the second, or remaining, eye. From the standpoint of etiology, reports on eyes rather than on patients may be misleading, causing the frequency of many diseases to seem greater than it actually is; e. g., in a case of syphilitic interstitial keratitis of both eyes syphilis would be twice designated as the cause of blindness, although there would be only 1 case of syphilis. In the reports from Pennsylvania and Missouri this discrepancy seems not to have been clearly pointed out. In future statistics, with the adequate data which are anticipated, reports will be on the basis of blind patients and blind eyes.

The Standard Classified Nomenclature of Disease is used for coding because of its vastly broader field of application, which permits the

coding of all conditions contained in the reports, whether the patient is blind or not. In its present form this nomenclature is difficult to use, but it is flexible, so that deficiencies can be filled in with numbers devised as needed. This coding saved a great deal of labor in making the etiologic table, which contains the diagnosis of the primary disease only—that is, the essential cause of blindness. Secondary or complicating conditions are not included, except in several instances in which cataract or glaucoma followed injury.

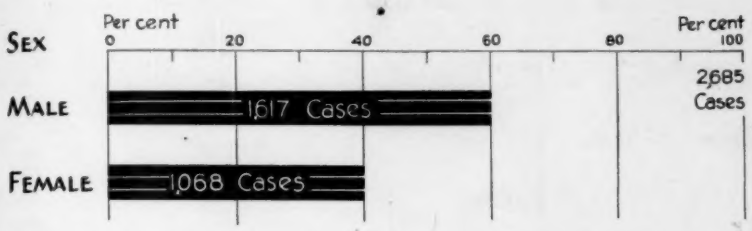

Chart 1.—Distribution according to sex of recipients of assistance registered between July 1937 and July 1939.

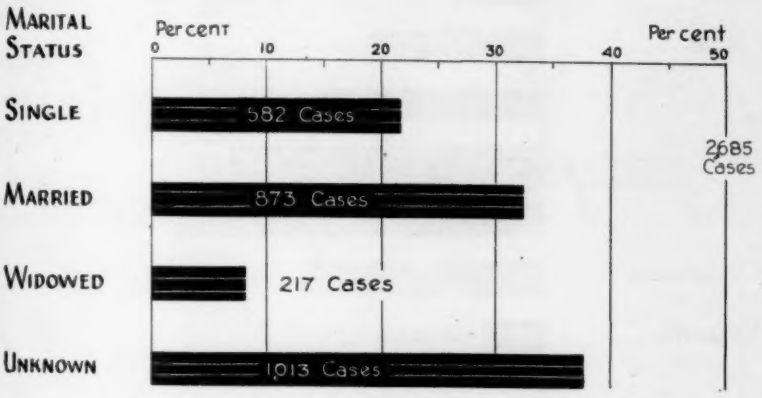

Chart 2.—Distribution according to marital status.

Of the 2,685 blind persons observed in the two year period, 1,617, or 60 per cent, were males, and 1,068, or 40 per cent, females (chart 1). This greater incidence of blindness among men is greater than the disproportion of males to females in the population of the state and is characteristic of all reports on blindness.

A study of the marital status (chart 2) revealed that of the 62 per cent for which this was indicated, 52 per cent were married, 13 per cent widowed and 35 per cent single.

Blindness is shown to be predominantly an affliction of advanced age (chart 3). Seventy-five per cent of all patients were past 40 years of age; 60 per cent were past 50 years, and 25 per cent were in the seventh decade of life. These figures must be qualified to a degree by the consideration of the greater need for assistance of patients in these age groups, which would induce greater numbers of them to register with the department.

According to the data on age at onset of ocular disease, 10 per cent were under the age of 1 year (chart 4). Hereditary and congenital

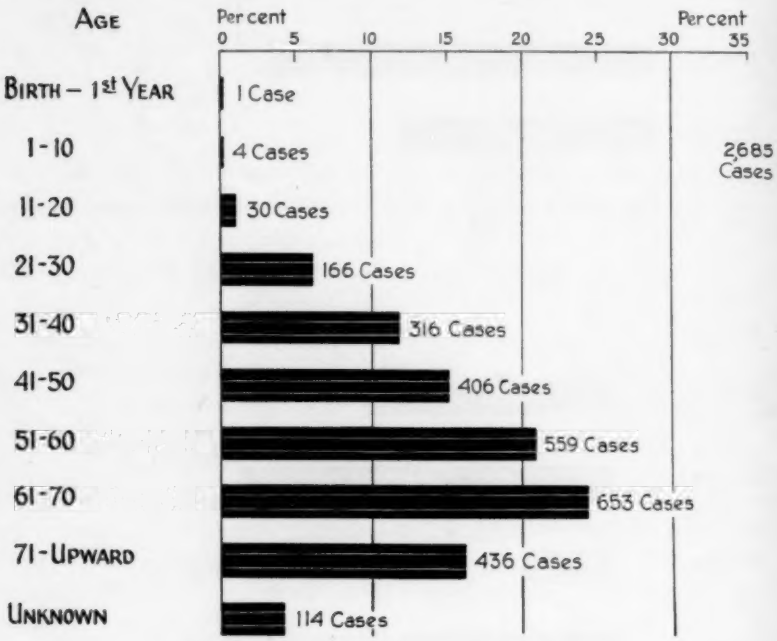

Chart 3.—Distribution according to age.

diseases and anomalies and ophthalmia neonatorum were the chief causes. In future statistics this age group percentage should gradually decline as a result of the present excellent control of the last-named disease, but a still too high figure for preventable blindness will remain. It is to be hoped that education will be effective in reducing it and that there will be no need for legislative measures.

With respect to the degree of blindness, 30 per cent of the subjects were found to be totally blind; 22.5 per cent had light perception; 46 per cent had 20/200 vision or less, and 1.5 per cent had 20/200 vision or better (chart 5). The last-mentioned percentage represents

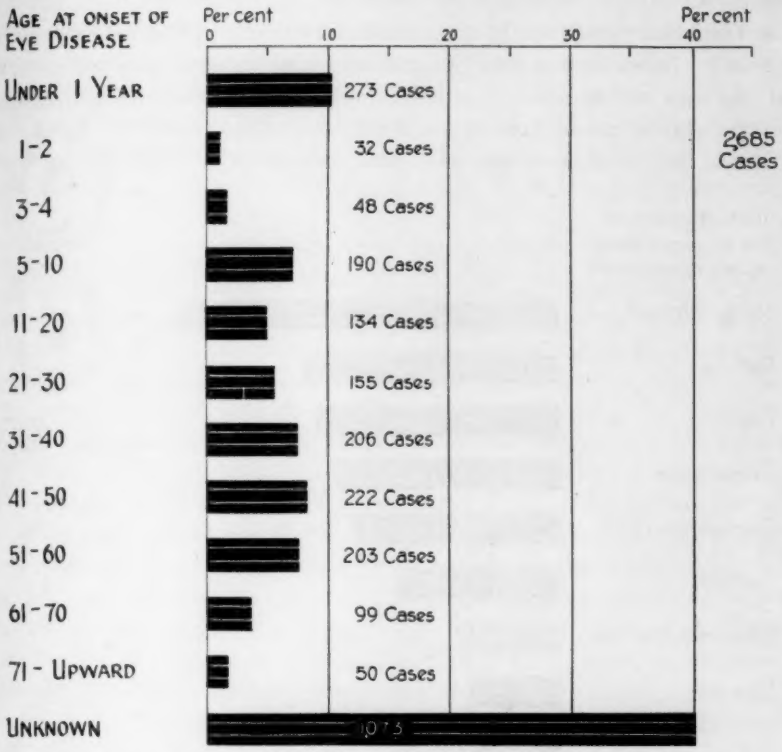

Chart 4.—Distribution according to age at onset of ocular disease.

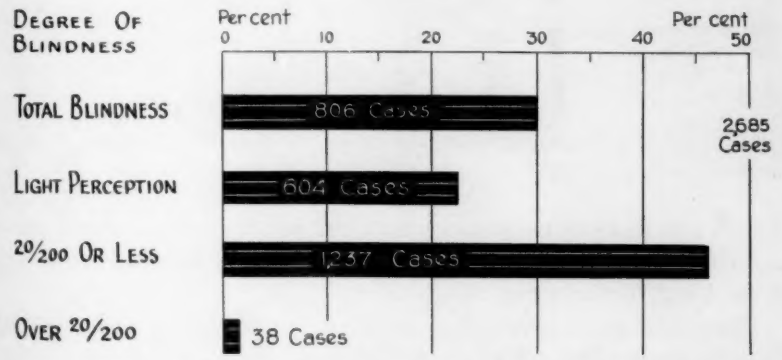

Chart 5.—Distribution according to degree of blindness.

38 persons who had visual field defects in the better eye of sufficient degree to justify the rating of "blind."

The blind population of the various counties receiving assistance was studied. It was noted that one-half the total number of blind persons of the state reside in the Greater New York area, which has one half of the state's population. The total percentage receiving assistance (2,685) of the total number of blind persons registered in the state

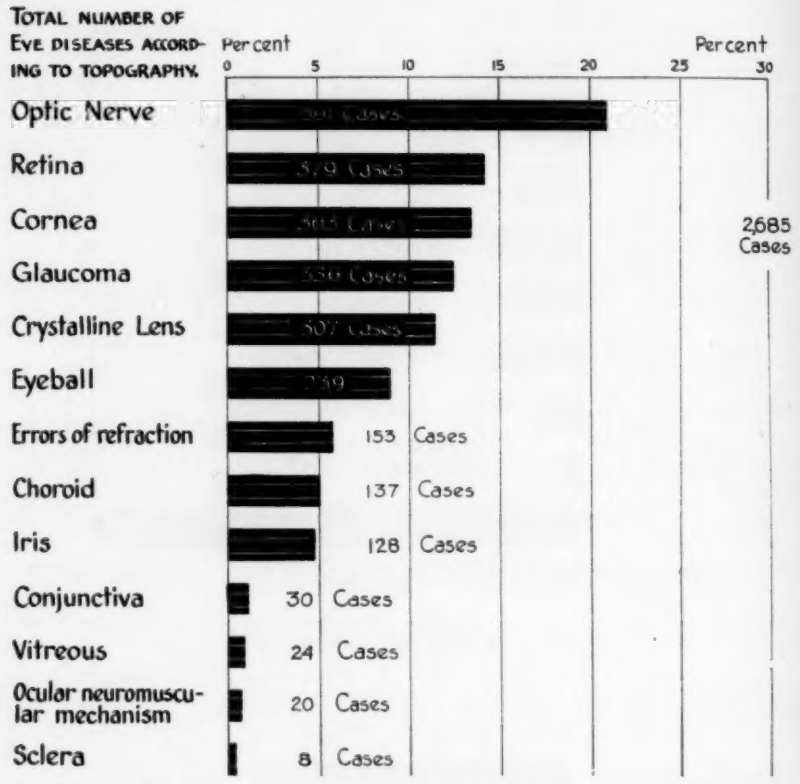

Chart 6.—Distribution according to causes of blindness and sites of ocular disease.

(14,692) is 18.25. This represents 00.002 per cent of the state's population (based on the 1930 census), which reduces somewhat the ratio of the blind to the seeing.

According to location, diseases of the optic nerve accounted for blindness in one fifth (561) of all the cases (chart 6). Of this number there were 512 cases of atrophy of this nerve, in 135 of which the condition was due to syphilis. Diseases of the retina produced blindness in 379 recipients, of whom 103 had retinitis pigmentosa and 93 had

detachment of the retina. Blindness due to diseases of the cornea occurred in 353 cases, in 270 of which it was attributed to leukoma of the cornea, which is an inadequate diagnosis. Glaucoma was the fourth most prevalent cause of blindness, occurring in 336 persons, and diseases of the lens resulted in blindness in 307 persons.

Treatment which might result in improvement of vision and consequently remove recipients from the relief rolls was indicated in 163 cases of cataract (133 of them surgical) and in 86 cases of glaucoma.

CONCLUSION

Statistics on blindness in the United States are incomplete. There is not, therefore, an accurate conception of the prevalence or causes of blindness. Few reports have been issued, and all of these relate the difficulty of compiling accurate data. This is due to the failure of a great many of the reports of individual cases of blindness to convey a precise understanding of the ocular conditions producing blindness. The study of blindness in the state of New York reported here is greatly qualified by this difficulty and is the basis of a plea for better reporting on the blind and the partially blind in the state of New York.

In the two year period from July 1937, when the Social Security Act became effective, to July 1939, 2,685 cases of blindness were reported. According to the data on age of onset 10 per cent of the patients were under 1 year of age. Hereditary and congenital diseases, anomalies and ophthalmia neonatorum were the chief causes. Seventy-five per cent of all patients were past 40 years of age, and 60 per cent were males. Diseases of the optic nerve accounted for 561, or 20 per cent, of all cases of blindness, and 135 of these were syphilitic. Diseases of the retina produced blindness in 379, or 14 per cent. Diseases of the cornea led to blindness in 13.5 per cent, and 270 of the 363 cases were attributed to "leukoma" of the cornea. Glaucoma was the fourth most prevalent cause of blindness (336 cases, or 12.5 per cent), and diseases of the lens resulted in blindness in 307 cases, or 11.4 per cent.

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MUSCLE BALANCE DETERMINATIONS AT THE READING DISTANCE

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OMAHA

It is the purpose of this paper to review some of the physiologic laws involved in ocular convergence as a basis for offering constructive criticism of some prevalent methods employed in determining ocular efficiency at the reading distance. The subject matter of this discourse is such that any ophthalmologist may verify the conclusions by means of the usual office equipment.

RELATION OF ACCOMMODATION TO CONVERGENCE

A fallacy which has existed for many years presupposes that a direct relation exists between accommodation and convergence, at a ratio of 1 D. of accommodation to 1 meter angle of convergence. Ever since the time of Donders one writer has copied from another and advised that the spherical correction should be adjusted to compensate for any existing esophoria or exophoria. The old masters seemed to lack definite knowledge of the value of accommodative convergence and of converging power as computed by the use of prisms. One shudders at the untold discomfort that has been caused by prescribing lenses modified on the assumption that excess plus power (or decreased minus power) would reduce the ill effects of esophoria or that an overcorrection of minus lenses would compensate for insufficiency of converging power. Those who, having due regard for the science of physiologic optics, first ascertain the correct lenses for the eyes and then measure the converging power by use of prisms are eminently rewarded by the results.

EXOPHORIA FOR NEAR VISION

It is not unusual for an ophthalmologist to regard exophoria for near vision as a criterion of convergence efficiency. One may determine approximately what value can be awarded to exophoria for near vision. In order to attain binocular single vision at 33.3 cm. (interpupillary distance, 60 mm.) it is necessary for each visual axis to turn 9 prism diopters inward from parallelism, a total of 18 prism diopters for the two eyes. With the object of fixation placed at a distance of 33.3 cm. and diplopia artifically produced by means of a 6 diopter prism placed base down over one eye (dissociation) suppose that a 5 diopter prism

base in is required to aline the two images vertically. Five prism diopters is thus the amount of exophoria for near vision and is the portion of the total convergence which is furnished by the fusion faculty. It indicates that with fusion rendered impossible by dissociation (prism base down over one eye) the visual axes have failed by 5 prism diopters to converge the required 18 prism diopters. However, the exophoria for near vision can in no wise be considered a criterion for measuring convergence efficiency, because if there is an adequate reserve of fusional power (16 to 20 prism diopters) over and above that required to maintain binocular singular vision, it is safe to assume that no convergence deficiency is present.

The difference between the total amount of convergence (18 prism diopters) and the exophoria for near vision (assuming orthophoria for distance) must be attributed to the influence of accommodation and to the proximity of the object. For example, if there is exophoria for near vision of 5 prism diopters and total convergence of 18 prism diopters, there will remain 13 prism diopters of convergence yet to be accounted for.

The calculations just made were based on the assumption of orthophoria at infinity. This is not always accurate, because a certain amount of exophoria or esophoria for distance is frequent. When the eyes converge for close work, the position of the visual axes (phoria) which was demonstrated by the dissociation tests at distance must be allowed for; therefore, instead of calculating the total convergence from parallelism one must add the exophoria at distance or subtract the esophoria, if either exists. For example, if there is exophoria of 3 prism diopters at distance, that amount of imbalance would have to be overcome in converging at the reading distance, giving a total of 21 prism diopters. Conversely, esophoria of 3 prism diopters would diminish the necessary convergence to 15 prism diopters.

Exophoria or esophoria for near vision simply indicates the position or direction of the visual axes when the influence of the fusion faculty is removed by prism dissociation. Since the fusion faculty is the dominant factor in preventing diplopia, it is evident that it must be taken into consideration in the determination of convergence efficiency. The efficiency of the fusion faculty may be measured by the strength of base out prisms which can be overcome without the loss of binocular single vision.

ACCOMMODATIVE AND PROXIMAL CONVERGENCE

Returning to the problem of further analyzing convergence, a pair of + 3 D. spheres under the dissociation is added to relax accommodation. Any residual convergence present under these conditions cannot be due

to fusion or to accommodation, since each eye sees a separate image from the effect of the prism (dissociation), while the spheres eliminate accommodation.

If the vertical images remain laterally separated (not vertically alined), it is reasonable to conclude that it is the psychic effect of nearness that induces the remaining convergence. Assuming (for illustration) that after the plus spheres have been added the images are separated to such an extent that the exophoria for near vision is increased to 12 prism diopters, there will still be left 6 (18 — 12) prism diopters of convergence, which is attributed to the psychic effect of nearness of the object (proximal convergence).

In the preceding paragraphs attention was drawn to the fact that the visual axes converge 18 prism diopters to fixate a test object at 33.3 cm. With fusion eliminated, the visual axes converged only 13 prism diopters. When accommodation was removed by the plus spheres, 7 prism diopters more of convergence disappeared, leaving the final 6 prism diopters to be attributed to proximity. Thus, it is seen that the factors which may be present in producing convergence at near vision can be divided into fusional, accommodative and proximal.

Accommodative convergence varies in normal persons from zero to the total amount of convergence necessary for close work. The question arises as to the part accommodative convergence normally plays in binocular single vision. There is little doubt that normally the faculty of fusion is the dominant factor in convergence. At birth there is no relation between accommodation and convergence. The accommodative convergence reflex is the result of a learning process, and the degree of its development has no bearing on ocular efficiency or comfort. Low accommodative convergence simply indicates a poorly established relation between accommodation and convergence. That such a condition produces no symptoms is proved in everyday practice; one constantly encounters low accommodative convergence in persons who are perfectly comfortable. The near phoria merely enables the ophthalmologist to compute the position of the visual axes when the influence of the fusion faculty has been removed and cannot be a criterion in the determination of convergence efficiency at the reading distance.

In making calculations for the surgical correction of strabismus, some of the best authorities use the near point of convergence as one criterion in deciding on the procedure to be used. For every movement of the eyeball there must be a stimulus which prompts the response. In cases of strabismus, when the test object is brought toward the eye it seems safe to assume that the version produced is accommodative and proximal convergence. If it is true that the amount of accommodative convergence

does not affect the comfort of the patient, it remains for the surgeon, who depends in part on this finding for deciding on his operative procedure, to explain the physiologic basis of the method used.

NEAR POINT OF CONVERGENCE

Determination of this point, which is almost universally used in refractive procedures, is made by requiring the patient to fixate a small test object which is brought toward him in the median line while the operator takes note of the point at which one eye fails to fixate. It is usually assumed that convergence efficiency is measured by this method. However, a little investigation will disclose sources of possible error which almost entirely abrogate the value of the findings.

In the first place, it is common to find cases in which the near point is close to the eyes and yet, when base-out prism power is placed at the reading distance, convergence is found to be deficient. In view of the fact that reading matter or other close work is usually held 38 to 40 cm. from the eyes, it seems consistent to make the determinations at that distance. Especially is this true since disturbing factors which may well influence the position of the near point come into play as the test object approaches the nose. The image on the retina becomes larger as the distance between the object and the nodal point of the eye shortens. As a person grows older the near point of accommodation recedes, with the result that a blurred image discourages fusion and diplopia ensues sooner than would be the case if the image were sharply defined. No one seems to have offered a scale of measurement based on the position of the near point of convergence which can be adapted to the age of the patient, the converging power at distance or a system of treatment. The worst objection of all is that the correct diagnosis would be missed entirely in many cases if the location of the near point of convergence were the only means used to determine convergence efficiency at the reading distance.

BOECK'S SARCOID

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The importance of submitting all excised tissue to the pathologist for careful examination is best exemplified by the following case.

REPORT OF A CASE

S. N., a woman aged 49, came to the office on May 3, 1939, because of a small cyst high on the left upper lid, in its inner half. The cyst presented all the appearances of a chalazion, and the patient was advised to have it removed.

She returned on June 10, when it was noted that the cyst had grown much larger. It was fairly firm and was freely movable except at its nasal end. I thought that the tumor was a fibroma, and the patient was admitted to the Brooklyn Eye and Ear Hospital on June 12.

With the region under local anesthesia a horizontal incision of the lid just below the supraorbital margin was made. Beneath the skin there was a somewhat flattened tumor which passed back into the orbit. This was excised.

Dr. J. Arnold de Veer, the pathologist, reported the mass to be a tuberculoma.

After the report a thorough examination of the patient (including roentgenograms of the chest) was made, with entirely negative results. Mantoux tests with increasing dilutions of tuberculin also gave negative results.

Shortly after the report Dr. de Veer stated that after reexamining the specimen he felt that a diagnosis of Boeck's sarcoid would be more accurate. His reasons for the change of diagnosis were as follows:

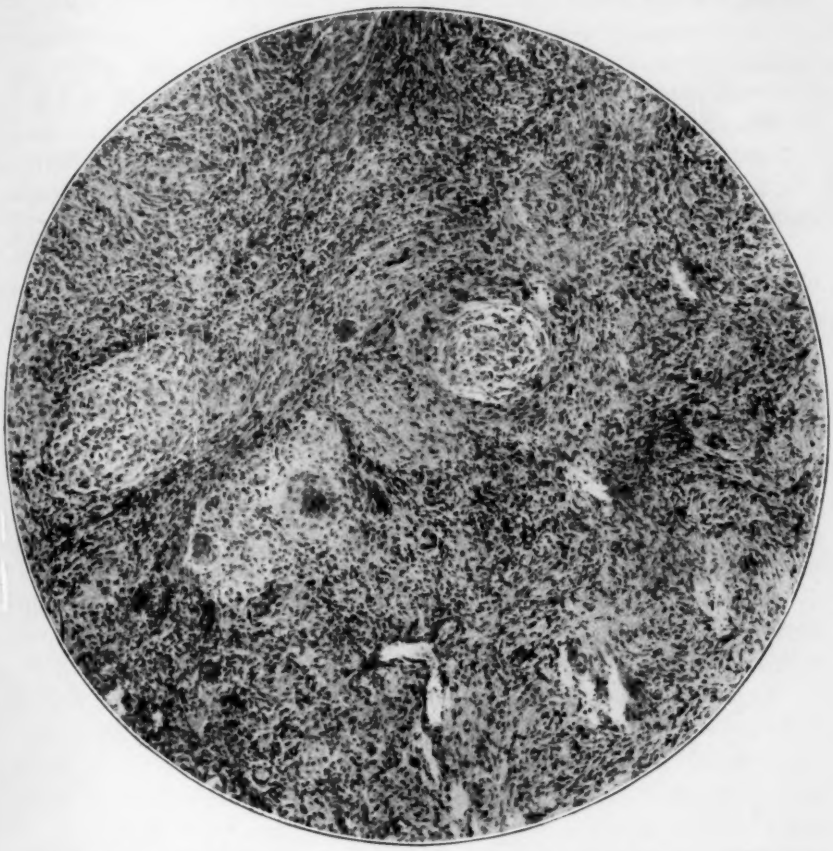
"The lesion was roughly ovoid and measured 1 by 2 cm. in its major diameter. It was firm, rubbery and grayish white, its appearance being not unlike that of scar tissue. Microscopic sections stained with hemotoxylin and eosin showed a structure that was at first considered to be that of atypical tuberculosis. Stains for acid-fast bacilli were made, but no such organisms could be demonstrated. Further examination revealed the following features, which in general are usually found in the lesion of Boeck's sarcoid:

"The predominant structures seen were numerous round or ovoid sharply circumscribed tubercle-like nodules, some containing no giant cells. No caseation necrosis was seen in any of these nodules. Narrow mantles of lymphocytes surrounded some, but most of the epithelioid lesions were devoid of any lymphocytic zone. There were patches of dense fibrotic tissue in which remains of the epithelioid structures persisted among collagenous material. These were evidently healed or healing portions of the lesion. No animal inoculations were made."

COMMENT

The clinical appearance of the patient was so far from suggesting such a condition that I was stimulated to investigate the literature. I was particularly interested in the relation between tuberculosis and Boeck's sarcoid.

In 1889 Boeck [1] described the case of a man aged 36 who was in good health except for a disease which first appeared on the brow and spread to the face, gradually extending onto the scalp, trunk and limbs. It consisted of a number of spots and patches the appearance of which varied with their stage of development. In size they varied from that of a small pea to that of a large bean. They were slightly elevated above the surface of the skin. They formed well defined nodules. The



Low power photomicrograph showing the granulomatous lesion, including a cluster of small epithelioid tubercles. One contains a number of giant cells.

swollen ones were uniformly yellowish brown and sometimes slightly scaling. Somewhat larger nodules showed a slight central depression of a bluish red tint sharply contrasting with the border. On close inspection a network of dilated capillaries was seen in the central area. Where the lesions had disappeared, dark brown atrophic spots remained.

1. Boeck, C.: Multiple Benign Sarcoid of the Skin, J. Cutan. Dis. **17**:543, 1899.

The cubital lymph nodes were enormously swollen. The femoral nodes were also enlarged, but the submaxillary and the cervical nodes were not. The patient was cured after a long period of treatment with arsenic compounds, but atrophic white and brown spots remained.

The new growths were described as composed of perivascular sarcomatoid tissue built up by excessively rapid proliferation of epithelioid connective tissue cells in the perivascular lymph spaces. Boeck concluded that the lesion is due to the tubercle bacillus.

In an excellent review of sarcoids and related lesions, Goeckerman [2] stated that there is no sharp dividing line between Boeck's sarcoid and the three other entities classified by Darier [3] and called by Kyrle tuberculosis cutis (lupus pernio, Darier-Roussy sarcoid and erythema induratum). He stated that, though the clinical pictures appear different, the histologic changes resemble one another so closely that microscopic differentiation is impossible.

Darier [3] as well as Goeckerman expressed the belief that these conditions are tuberculous in origin and that they differ only because of the differences in individual reactivity.

Kyrle [4] reported the case of a man aged 22 with almost universal involvement and stated the opinion that the condition is a form of tuberculosis of the skin. He demonstrated the bacillus of tuberculosis in early lesions of Boeck's sarcoid, and according to him the cutaneous lesions are a foreign body reaction to the tubercle bacillus and its distribution products. Later only a few bacilli were found, and still later none were demonstrable, though by this time typical lesions of sarcoid were present.

Lewandowsky [5] injected living bacilli subcutaneously into partially immunized animals and failed to find tubercle bacilli, though the local lesions persisted for months.

Kyrle explained this by assuming that the skin reacts to the foreign bodies. If the bacilli proliferate actively a severe inflammatory reaction results. Patients with sarcoid sometimes give a history of a severe inflammatory condition. If, however, the bacilli are inactive, a tuberculid reaction occurs.

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2. Goeckerman, W. H.: Sarcoids and Related Lesions, Arch. Dermat. & Syph. **18**:237 (Aug.) 1928.

3. Darier, J.: Die cutanen und subcutanen Sarkoide, Monatsh. f. prakt. Dermat. **50**:419, 1910.

4. Kyrle, J.: Die Anfangstadien des Boeckschen Lupoids; Beitrag zur Frage der tuberkulösen Aetiologie dieser Dermatose, Arch. f. Dermat. u. Syph. **131**:33, 1921.

5. Lewandowsky, F.: (*a*) Tuberculose- Immunität und Tuberkulide, Arch. f. Dermat. u. Syph. **123**:1, 1916; (*b*) Zur Kenntnis der Boeckschen Sarkoide, ibid. **135**:287, 1921.

Six of the patients reported on by Goeckerman had tuberculous lesions. Sulzberger said that Boeck's sarcoid must be regarded as a form of cutaneous tuberculosis. He concluded that any other view would be a decided step backward.

Osterberg [6] reviewed 500 cases of Boeck's sarcoid, among which were 27 cases of iritis. One of the patients had a positive tuberculin reaction which persisted after spontaneous healing of the lesion.

Folk [7] reported the case of a man aged 49 who had a 2 plus Mantoux reaction.

Those who believe Boeck's sarcoid to be a form of tuberculosis explain the usually negative tuberculin reactions and the absence of tubercle bacilli in the lesions, as well as the failure to cause tuberculosis in susceptible animals by inoculating them with sarcoid tissue, as due to a state of anergy, which is a form of immunity.

Walsh [8] linked sarcoid to uveoparotid fever and stated that both conditions are probably forms of tuberculosis.

Sulzberger [9] stated that in the process of immunity hyposensitivity is often achieved via hypersensitivity, and he claimed to have seen patients with sarcoid who were extremely sensitive to tuberculosis.

Goeckerman, however, pointed out that many excellent observers do not believe that all sarcoids are tuberculous in origin. In fact, some believe that each of the sarcoids is due to a different cause, in spite of the similar clinical and histologic pictures.

Blegvad [10] reported 3 cases in which there was involvement of the conjunctiva as well as of other parts of the body. Animal inoculations as well as staining for tubercle bacilli gave negative results. He contended strongly against the idea that Boeck's sarcoid is a form of tuberculosis and differed sharply with those who attempt to explain the negative tuberculin reactions and the negative results of animal inoculations as due to a form of anergy.

Harrison [11] expressed the belief that Boeck's sarcoid is a granuloma. In fact, Morse and Stott [12] showed that a boiled alcoholic extract

6. Osterberg, G.: Boeck's Sarcoid, Brit. J. Ophth. **23**:145, 1939.

7. Folk, M. R.: Boeck's Sarcoid of the Palpebral Conjunctiva, Arch. Ophth. **24**:462 (Sept.) 1940.

8. Walsh, F. B.: Ocular Importance of Sarcoid, Arch. Ophth. **21**:421 (March) 1939.

9. Sulzberger, M. B.: Sarcoid of Boeck and Tuberculin Anergy, Am. Rev. Tuberc. **28**:734, 1933.

10. Blegvad, O.: Boeck's Sarcoid of the Conjunctiva, Acta ophth. **9**:180, 1931.

11. Harrison, B. J. M.: A Textbook of Roentgenology, Baltimore, William Wood & Company, 1936, p. 591.

12. Morse, P. F., and Stott, E.: Studies of the Tissue Reactions to Various Products of the Tubercle Bacillus, J. Lab. & Clin. Med. **2**:159, 1916; cited by Goeckerman.[2]

of tubercle bacilli yielded a substance that produced a lesion similar to that produced by the germs, a foreign body reaction.

Derby and Verhoeff [13] reported the case of a man aged 25 who noticed a growth in the outer half of the right upper lid. It was yellow-red and felt cartilaginous. Three months later a nodule developed on the right cheek. Both were excised. Verhoeff, who examined the specimen, stated that the sharp definition of the tubercle and the lack of inflammatory reaction strongly suggested either a foreign body or an obscure metabolic reaction.

Stumpke [14] observed a case of Boeck's sarcoid in which search for the tubercle bacillus was fruitless.

Finnerud [15] reported the case of a woman aged 25. The condition began with swelling of a little finger and then spread over the entire body, with destruction of the distal ends of several fingers and toes. Animal inoculations as well as staining for the tubercle bacillus gave negative results for tuberculosis.

Nickerson [16] reported the autopsy observations in 6 cases of clinically undiagnosed Boeck's sarcoid. He called attention to the fact that sarcoid is seldom fatal even when associated with visceral involvement. Five of the patients died of other major diseases. The sixth was believed to present a rare example of fatal sarcoid. She was a Negress who had a tumor removed from an eyelid. The histologic diagnosis of the removed tissue was atypical tuberculosis with involvement of the lacrimal gland. There was no caseation, and no acid-fast bacilli were found in the lesion. Six months later she returned to the hospital very ill, and her condition was diagnosed as atypical tuberculosis.

At autopsy numerous flat nodules 1 by 4 cm. were found scattered over the parietal pleura of the right lung and over the parietal pericardium. The subsequent histologic diagnosis was sarcoid.

The most constant observation at autopsy in all the reported cases was splenomegaly with or without lymphadenopathy.

Nickerson opposed the theory that Boeck's sarcoid is a form of tuberculosis. He pointed out that the disease is a benign condition of middle life which runs a chronic course with spontaneous remissions.

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13. Derby, G., and Verhoeff, F. H.: Sarcoid of the Eyelid, Arch. Ophth. **46**:312, 1917.

14. Stumpke, G.: Zur Frage des Boeckschen Sarcoids, Med. Klin. **16**:178, 1920.

15. Finnerud, C. W.: A Clinical, Histologic and Bacteriologic Study of Multiple Benign Sarcoid of Boeck-Darier-Roussy, Arch. Dermat. & Syph. **4**:342 (Sept.) 1921.

16. Nickerson, D. A.: Autopsy Findings in Six Cases of Boeck's Sarcoid, Arch. Path. **24**:119 (July) 1937.

According to him, the following criteria are necessary to make a diagnosis of sarcoid and to differentiate it from tuberculosis.

1. Caseation is never present.

2. The giant cells are different from those associated with tuberculosis. They are usually larger; then they contain more nuclei, and the nuclei are more evenly distributed and are seldom arranged in the elliptic manner typical of tuberculous giant cells.

3. Lesions in tissues other than the lungs do not contain carbon pigment.

4. No evidence of tuberculosis is found elsewhere in the body.

CONCLUSION

It is still in dispute whether Boeck's sarcoid is a form of tuberculosis, a neoplastic disease (Spiegler-Fendt type of sarcoid) or the response to a toxemia, but it seems to me that the preponderance of clinical evidence is against a tuberculous origin.

It is difficult to conceive how such widespread lesions can heal spontaneously or under arsenic therapy when it is known how resistant to treatment are proved tuberculous cutaneous diseases (e. g., lupus erythematosis).

Pathologically it is difficult to link sarcoid with tuberculosis when caseation, negative results of animal inoculation tests and absence of tubercle bacilli are almost constant observations.

The case reported is unusual in that there was no cutaneous involvement and no tendency toward spreading of the lesion to other parts of the body.

It would therefore seem that sarcoid can vary from a widespread disease with a fatal termination to a disease so benign that its only manifestation is an isolated small tumor.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

PATHOLOGIC CONDITIONS OF THE CORNEA

II. THE SUBSTANTIA PROPRIA

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The initial half of this work [1] dealt with the epithelium, the endothelium and Bowman's and Descemet's membranes. It was pointed out that the primary characteristic of the cornea is transparency, which is dependent on the water content of the tissue, and that this is regulated by the anatomic and functional integrity of the delimiting layers. In this installment there will be viewed the behavior of the substantia propria, which forms the bulk of the corneal tissue.

ANATOMY

The substantia propria, or stroma, is composed of broad, thin lamellas arranged parallel to the surface of the cornea and lacing themselves together in an in and out manner. Each lamella is composed of fine connective tissue fibrillae arranged in a parallel direction. Elastic tissue fibers are present throughout but are especially numerous posteriorly. It is this parallelism of the structural elements that plays an important role in the transparency of the tissue.

Cells are also present in the stroma; these are (*a*) fixed and (*b*) wandering. The fixed (connective tissue) cells lie in the lamellar interspaces and because of this compressed position have a flattened appearance. The wandering cells are leukocytes, with their lobulated nuclei also compressed by their constricted environment.

There are no blood vessels in the normal corneal stroma. Nerves, however, are abundant. These enter at the periphery and branch off, forming a plexus in the middle and anterior layers and sending fibrillae through Bowman's membrane to the epithelium.

GENERAL PATHOLOGIC CONDUCT

The reactions of the cornea to disease are largely influenced by its avascularity. Its nutrition is served chiefly by a process of diffusion

From the Registry of Ophthalmic Pathology, Army Medical Museum.

1. Rones, B.: Pathologic Conditions of the Cornea, Arch. Ophth. **24**:374 (Aug.) 1940.

of the fluids from the vascular plexus around the limbus and also indirectly from the aqueous. There are no fixed channels for diffusion of this fluid. The metabolism of the stroma is of an extremely low order, which considerably affects its behavior. The cornea is not particularly liable to involvement in systemic infectious diseases, but it has a greater proclivity to partake in allergic reactions. Owing to its slow metabolism, the acquired corneal hypersensitivity is prolonged, and this makes it capable of allergic response to attenuated toxic processes which do not involve tissues with a higher metabolic rate.[2]

The transparency of the cornea is dependent partly on its anatomic structure and partly on the fact that the corneal elements have nearly the same index of refraction.[3] Localized changes of this index of refraction produce opacities, even though such changes are difficult to detect on histologic examination. When the normal amount of corneal fluid is increased, the tissue swells and turns opaque. Edema so caused can follow diverse conditions—trauma, inflammation, degeneration or glaucoma.

Since the lamellas of the stroma are arranged in layers, it is easier for inflammatory cells and blood vessels to invade the cornea between the layers than through them. This produces the characteristic pictures that will be discussed in which both inflammations and vascularizations occur in the superficial stroma or in the deeper layers, depending on the points of greatest response to the toxic agent.

PIGMENTATION

Hematogenous pigmentation of the stroma is frequently seen with large hemorrhages into the anterior chamber, particularly those associated with increased intraocular tension. This staining is not due to the intact red blood corpuscles wandering into the stroma but is resultant on the absorption through Descemet's membrane of their split products, especially hemosiderin. The pigmented granules are seen in the corneal corpuscles and in the lamellar interspaces.

Hemorrhages can occur in the stroma, however, from the rupture of an invading blood vessel. In this case the intact red blood cells will be seen to separate widely and to mask the lamellas.

DEGENERATION

The most common degenerative change seen in the corneal stroma is arcus senilis. Takayasu [4] demonstrated this to be due to an infiltra-

2. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2.

3. Friedenwald, J. S.: The Pathology of the Eye, New York, The Macmillan Company, 1929.

4. Takayasu: Beiträge zur pathologischen Anatomie des Arcus senilis, Arch. f. Augenh. **43**:154, 1901.

tion of fat droplets. These are present both in the lamellas and between them. They are contained in the corneal corpuscles and in large foamy histiocytes, which are present in all the layers. The heaviest infiltration is in the stroma beneath Bowman's membrane and anterior to Descemet's membrane.

Other conditions of corneal lipoidosis have been described. Heath[5] reported a case of deep clouding of the cornea with vascularization. Smears made from needle punctures showed small lymphocytes, extra-

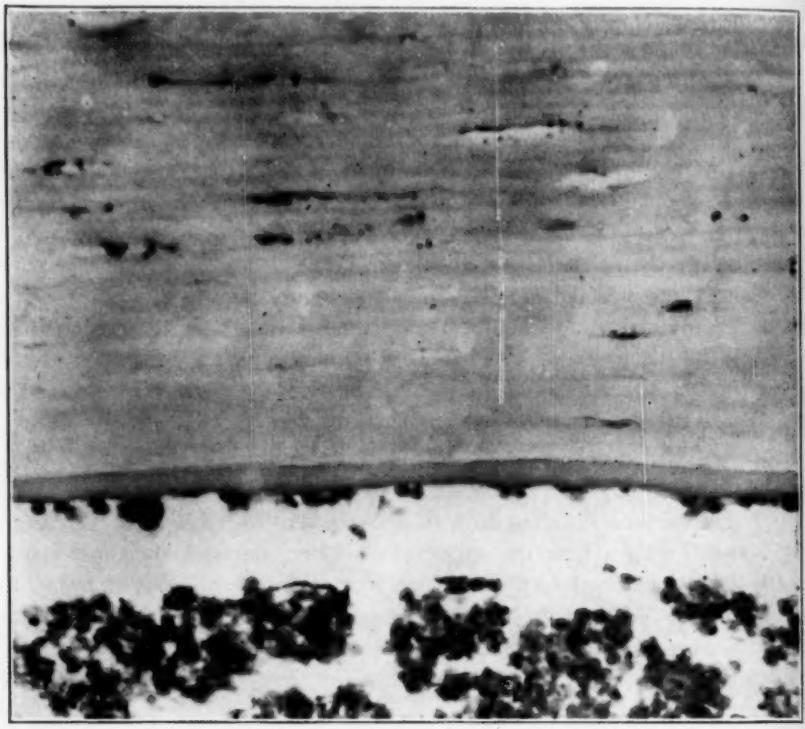


Fig. 1. (Army Medical Museum negative 71046).—Hematogenous pigmentation of the cornea from hemorrhage into the anterior chamber.

cellular fat crystals and large pale histiocytes storing fat. Berliner[6] described a case of "lipin keratitis" associated with Hurler's syndrome. He found the lipid deposits to be lying in the dilated, spindle-shaped interlamellar spaces. Brown and Katz[7] fully discussed the condition

5. Heath, P.: Lipin Interstitial Keratitis, Arch. Ophth. **13**:614 (April) 1935.

6. Berliner, M. L.: Lipin Keratitis of Hurler's Syndrome (Gargoylism), Arch. Ophth. **22**:97 (July) 1939.

7. Brown, E. V. L., and Katz, D.: Dystrophia Adiposa Corneae, Tr. Am. Ophth. Soc. **30**:173, 1932.

called "dystrophia adiposa corneae." They concluded that the condition is brought about by an impairment of the physiologic processes of the corneal cells, so that fat brought to the cornea is not utilized and becomes deposited there. Impaired cell metabolism leads to the disintegration of the lamellas and the liberation of the "invisible fat," a component of which is cholesterol with its esters. The presence of these substances results in a chemical irritation which produces circumcorneal injection and corneal vascularization. The latter may occur for the dual purpose of furnishing direct nourishment as well as for transporting phagocytes into the pathologic area. In Brown and Katz's case there was a diffuse distribution of fat bodies of various size. The greatest

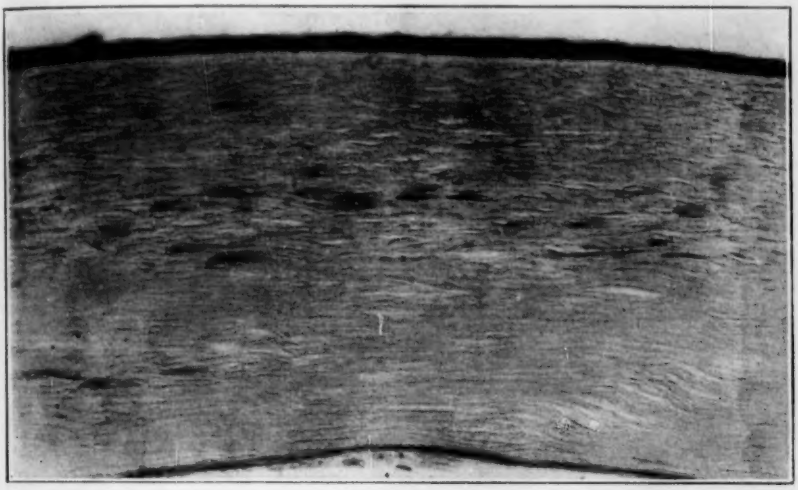


Fig. 2.—Necrosis of posterior corneal layers in a case of acute glaucoma. Note the absence of corpuscles in the posterior layers of the stroma. (Negative 71048.)

deposition of this fat was extracellular and occurred in the middle and posterior layers of the stroma. Fat-laden histiocytes were also present.

Another degenerative change, which shows nothing characteristic histologically, is marginal ulcer, or Mooren's ulcer. The condition is essentially a form of necrosis of the stroma and of the peripheral portion of Bowman's membrane. The deeper layers of the stroma become vascularized and infiltrated with lymphocytes. Granulation tissue develops in the ulcer, and the thickened epithelium grows to cover it.

WOUNDS AND REPAIR

In the initial paper it was educed that small wounds penetrating Bowman's membrane are repaired by the epithelium alone. If, however,

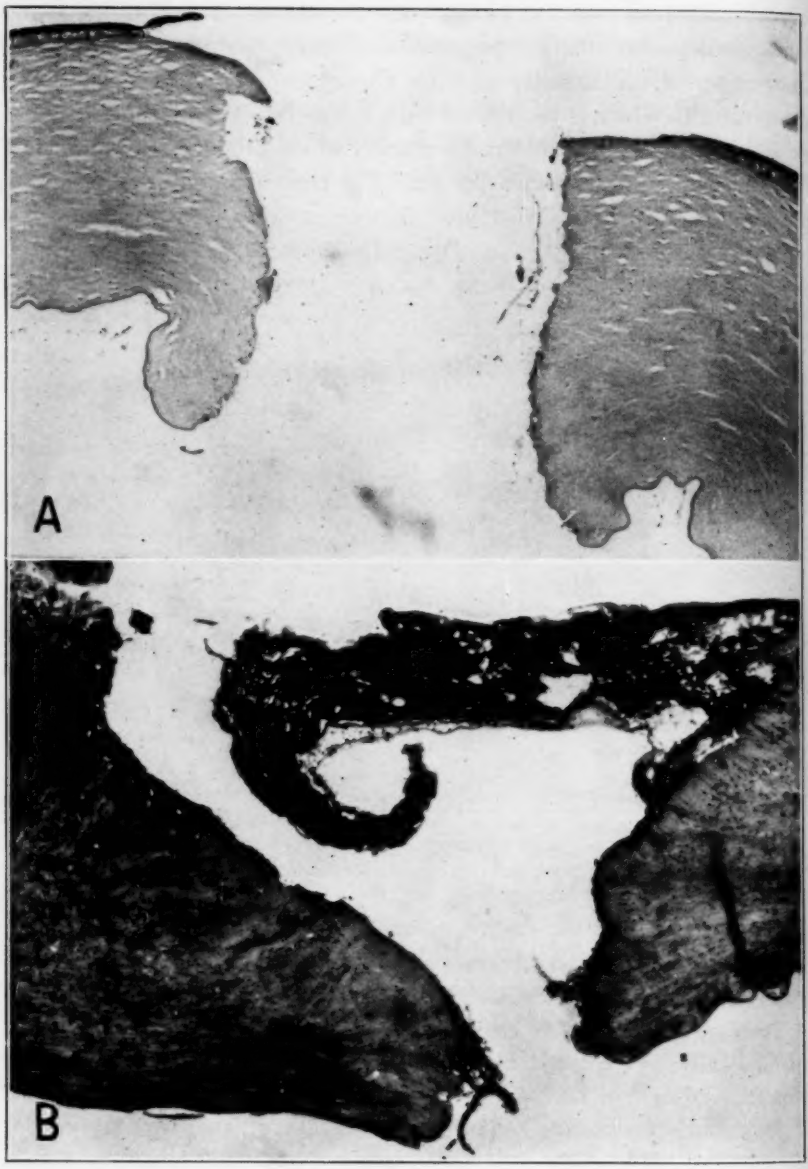


Fig. 3.—*A,* fresh perforation. The lips of the wound are edematous, but no cellular reaction has yet occurred. (Negative 71047.) *B,* perforated ulcer, partially filled with fibrinous exudate and disorganized epithelium. The stroma is infiltrated with polymorphonuclear leukocytes and lymphocytes and shows some vascularization. (Negative 71038.)

there has been a considerable loss of the substantia propria, the epithelium cannot in itself compensate for it even though it does grow over the area. There has been described a regeneration of the corneal tissue beneath this epithelium. Salzer [8] has detailed the appearance of

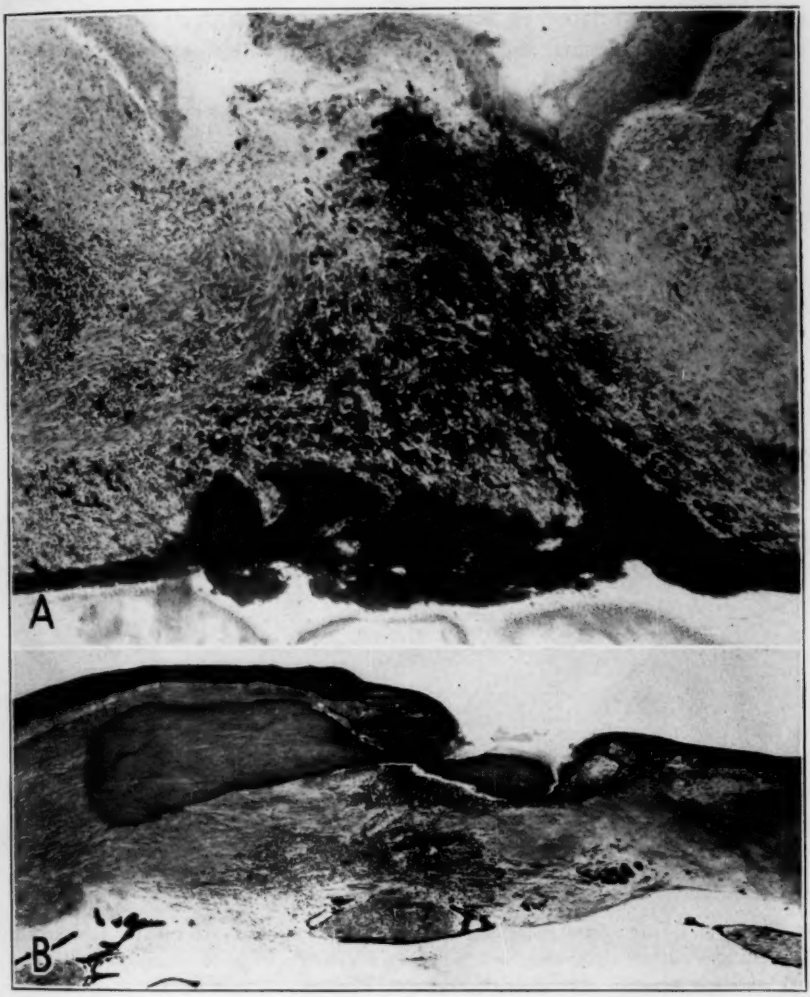


Fig. 4.—*A*, perforation of a gonorrheal ulcer, with incarceration and necrosis of the iris. (Negative 71049.) *B*, gonorrheal keratitis with recent perforation filled with plasma cells and purulent exudate and partially bridged by a calcified plaque of old scar tissue. (Negative 71070.)

8. Salzer: Weiteres über experimentelle Einheilung konservierter Hornhautsubstanz in die Hornhaut des Kaninchens, Arch. f. Augenh. **73**:109, 1913.

keratoblasts, which multiply by both mitotic and amitotic division and develop into fibers. These fibers tend gradually to assume the characteristics of the normal substantia, in that they become thicker and their arrangement more regular. When such small injuries occur in childhood, the corneal stroma can regain a considerable degree of transparency after the lapse of a long period. It is difficult to distinguish the tissue from normal tissue by histologic methods, except that Bow-

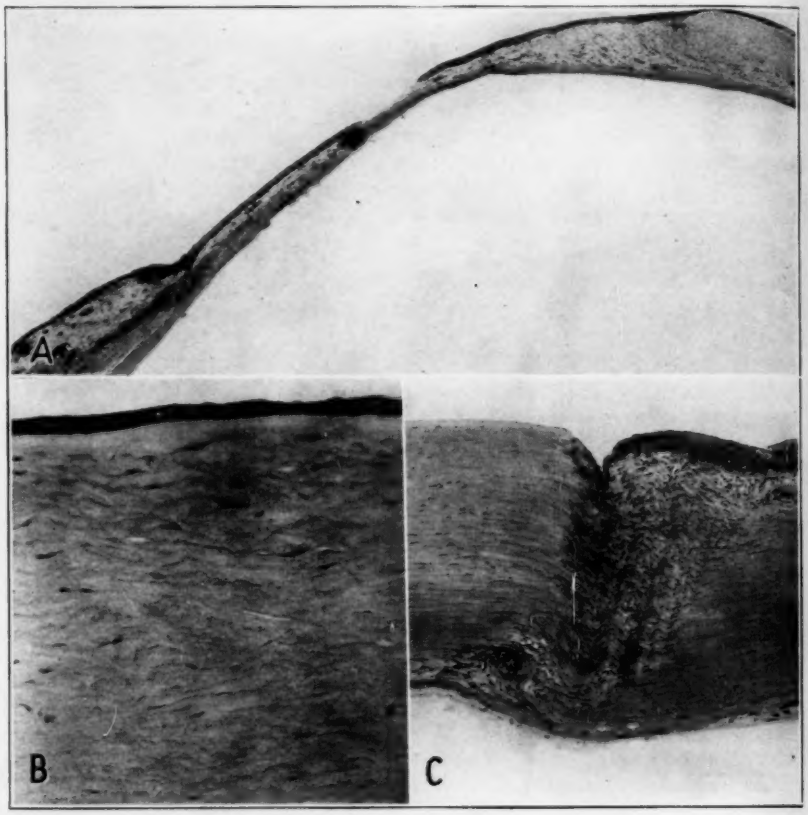


Fig. 5.—*A*, old gonorrheal keratitis. The cornea is irregularly thickened, scarred and partially vascularized. There is a localized area of marked thinning and necrosis beneath the ulcer. (Negative 71051.) *B*, old keratitis with dense scarring of the stroma. (Negative 71044.) *C*, recent scar at the limbus. The fibrous tissue is still loose and shows chronic inflammatory cell infiltration. (Negative 71069.)

man's membrane is absent. There has been voluminous dispute as to the origin of the keratoblasts, but they probably derive from proliferation of the fixed corneal corpuscles. The aforementioned march of events develops only in uncomplicated cases. When infection or vascularization

occurs, granulation tissue evolves from the invading blood vessels and fibroblasts, and this consolidates into scar tissue by hyalinization of the fibrous tissue and atrophy of the blood vessels.

An uncomplicated perforating wound rapidly begins to show an active reparatory process. The edges of the torn lamellas become swollen by the imbibed aqueous, so that they are quickly brought into apposition. Owing to the elasticity and retraction of Descemet's mem-

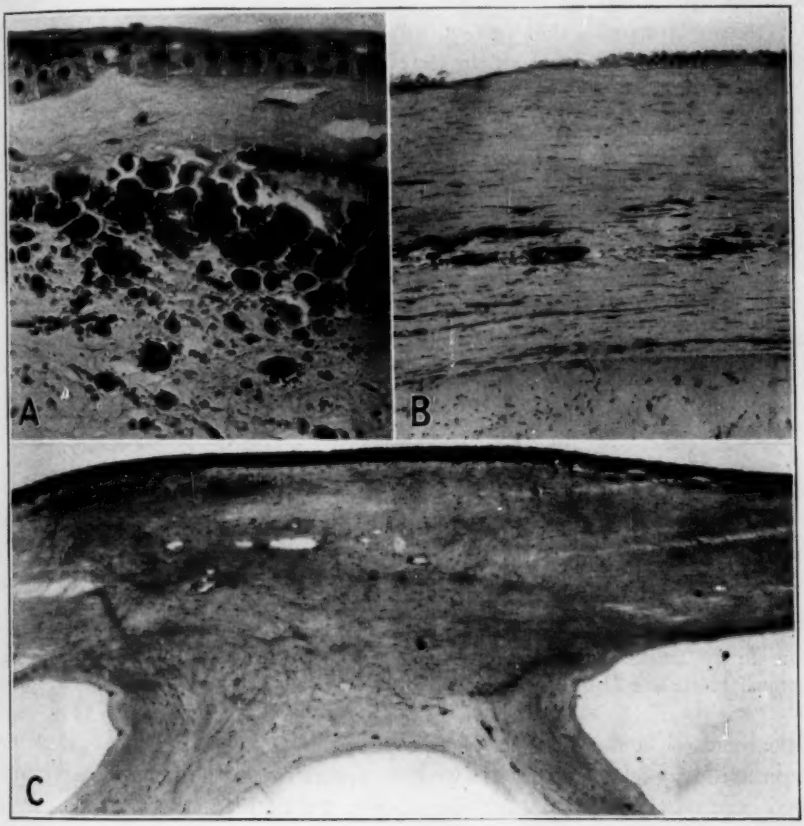


Fig. 6.—*A*, degeneration of a corneal scar. Numerous small granular and large amorphous degenerative products are present in the stroma. Bowman's membrane is fragmented and contains calcium deposits. (Negative 71040.) *B*, tattooing of a corneal scar. Pigment masses are scattered through the deep layers of the stroma. (Negative 71053.) *C*, scar of a perforating wound. A mass of scar tissue extends through the anterior chamber and is covered by a proliferating Descemet membrane. (Negative 71043.)

brane and the stroma adjacent to Bowman's membrane, apposition first occurs in the central area of the wound. This produces wedge-shaped gaps anteriorly and posteriorly, which become filled with fibrinous

coagulum and proliferated epithelium and endothelium. Later there occurs a considerable proliferation of keratoblasts, which develops into fibrous tissue fibrillae that use the coagulum as a scaffold to bind together the lamellas. The course of these newly formed fibers is irregular, but in time the scar shrinks, owing to their contraction and their tendency to become horizontal.

Frequently the rapid escape of aqueous through a perforating wound carries with it the iris or the lens to block the wound. This is beneficial in that it prevents the loss of the intraocular contents and bars the entrance of organisms from the exterior. If the iris is allowed to remain

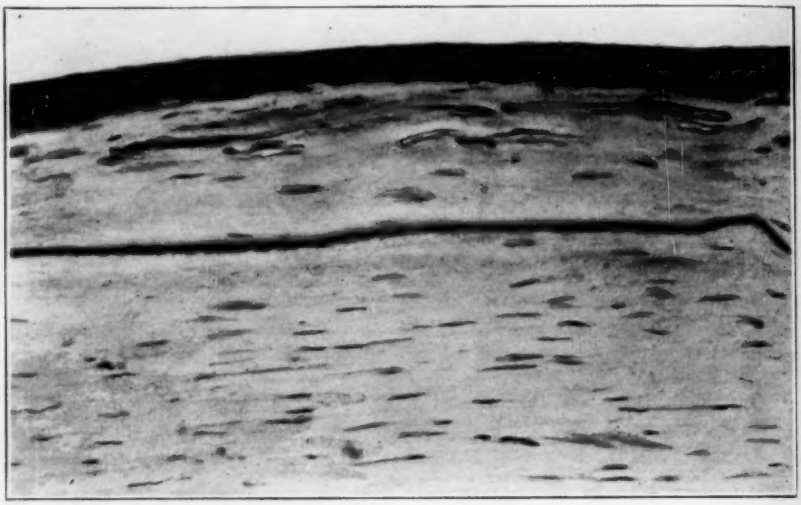


Fig. 7.—Calcareous plaque in a case of old keratitis. The calcium deposits are in the anterior part of the stroma beneath the fragmented Bowman's membrane. (Negative 71050.)

incarcerated, a fibrinous exudate rapidly binds it to the tissue, and the epithelium quickly covers the wound. Granulation and then scar tissue develop, so that the iris and the cornea are bound together in a dense scar, only the iris pigment being distinguishable. If the ruptured lens and its capsule also become incarcerated in the wound, healing is delayed and a violent inflammation is incited. The opaque and swollen lens fibers are invaded by fibroblasts, which bind the lens to the cornea by a mass of scar tissue. The vitreous and even the retina can be entangled in this mass and become adherent to the corneal scar. If the wound is not promptly sealed either by apposition or by prolapse of tissue, there may develop a fistulous tract through the cornea; this becomes lined by the downgrowth of epithelium and allows the constant seepage of

aqueous. Occasionally a perforating wound may be complicated by the inclusion of a cilium or some other foreign body, which, when retained, excites an inflammatory reaction, in which foreign body giant cells are a characteristic feature. Usually, however, the foreign body is expelled.

It may be stated that the deeper the destruction of corneal tissue the more dense and permanent is the scar. If the loss of substance has been extensive, the scar tissue may not be capable of repairing the defect. The thin corneal barrier will then not be able to withstand the intraocular pressure, and it will bulge. The resulting ectasia may continue to push forward until only Descemet's membrane is able to withstand the pressure, forming a descemetocele.

Degenerative changes occur in scars. Hyaline and amorphous substances are deposited in granules which coalesce and form large masses. Lipoid degeneration and calcification also develop in long-standing scars. At times calcareous plaques are desquamated, and the remaining stroma becomes covered with epithelium.

INFLAMMATION

Pathologists for many years have utilized the cornea as a favorite tissue to study the sequence of events in inflammation. This is due to the transparency and accessibility of the cornea. In spite of this, however, there is still considerable dispute as to the cells participating in the reaction. Since Bowman [9] in 1849 first described the proliferation of the corneal corpuscles, there has been disagreement as to their contribution. Marchand [10] demonstrated that they become contractile and capable of movement during inflammatory processes and that they are able to ingest foreign substances. They multiply and form large mononuclear histiocytes, which aid in the removal of necrotic tissue. These macrophages now appear as large round cells with large nuclei, which rapidly proliferate by division. Where the lamellas still persist, the macrophages are distorted and difficult to distinguish from cells of hematogenous origin. Lamb [11] studied the behavior of the corneal corpuscles in the reaction of hypersensitiveness. Starting with the normal thin, disk-shaped corpuscle with its dark-staining, large, round nucleus, it was first noted that the cells became larger and more clearly defined, with a thicker and more lightly stained nucleus. Division of

9. Bowman, W.: Lectures on the Parts Concerned in Operations on the Eye, and on the Structure of the Retina, London, Longman, Brown, Green & Longman, 1849.

10. Marchand, F., in Krehl, L., and Marchand, F.: Handbuch der allgemeinen Pathologie, Leipzig, S. Hirzel, 1924, vol. 4.

11. Lamb, H. D.: Corneal Corpuscles in the Reaction of Hypersensitiveness, Am. J. Ophth. **18**:644, 1935.

the enlarged corneal corpuscles was direct, and mitotic figures were not observed. Surrounding each focus of reaction simple proliferation was observed, but in the central necrotic zones the cells exhibited considerable degeneration and fragmentation of the nuclei and resembled pus cells.

Although the fixed corneal corpuscles play their role as macrophages in the processes of destruction of toxic substances and of repair, the leukocytes derived from the blood are far more important in the drama. As microphages they are able to phagocytose bacteria and neutralize bacterial toxins, besides aiding in the removal of necrotic tissue. These cells are drawn from the perilimbic plexus by chemotaxis, infiltrating into the stroma in response to the toxins which have diffused to the periphery from the site of injury or infection. The character of the responding cells depends on the nature of the stimulus. Thus, in purulent inflammations the predominant cells are the polymorphonuclear leukocytes, while in nonpurulent inflammations lymphocytes and plasma cells dominate. However, it is at times difficult to distinguish the cells, for they infiltrate between the lamellas and become compressed, thus losing their characteristic morphologic structure. Councilman [12] demonstrated that in eighteen to twenty-four hours after a central infection of the cornea with staphylococci leukocytes were present in the periphery and that only after four days did lymphocytes and plasma cells appear. This stage of infiltration manifests itself clinically as dulness and swelling of the cornea, which in cases of mild involvement returns to almost complete transparency by resorption of the infiltrate. When the process is severe and a considerable amount of corneal tissue becomes suppurative, ulcerative keratitis results.

Stokes,[13] in his study of the macrophages in experimental keratitis, has shown that the macrophages derived from the fixed cells of the cornea are identical with the macrophages of vascular origin observed when these cells are storing dye granules. Lacking the occasion for phagocytosis, however, the two cell types remain totally different. He found no evidence of the transformation of the cells of the corneal endothelium into macrophages.

PURULENT KERATITIS

Purulent inflammations of the cornea are most often secondary to trauma, for the normal corneal epithelium is resistant to all organisms except the gonococcus and the diphtheria bacillus. The organisms gain

12. Councilman, W. T., cited by Duke-Elder,[2] p. 1878.

13. Stokes, W. H.: Notes on Experimental Keratitis: An Investigation of the Cellular Pathology with Particular Reference to the Macrophages, Tr. Am. Ophth. Soc. **36**:316, 1938.

entrance to the stroma through the abrasion and produce tissue necrosis, partly from the action of the toxins and partly from the digestive action of the infiltrating leukocytes. The epithelium and Bowman's membrane are destroyed and desquamated, and the underlying stroma becomes densely infiltrated with pus cells. The necrotic stroma is then sloughed off, leaving a corneal ulcer. The infiltration may spread laterally, so that increasing areas of the cornea become ulcerated until complete necrosis results. It may also penetrate deeper into the cornea until Descemet's membrane is exposed; the destruction of this membrane will cause a perforation. Such a sequence of events may occur when the resistance of the tissue or of the patient is lowered. Usually, however, the defensive mechanism comes into action, forming a line of demarcation. The mobilized pus cells do not succumb to the toxins as in the central zone but are able to digest the necrotic tissue and clean up the

Fig. 8.—Purulent ulceration. The central ulcer shows considerable loss of stroma. The deeper layers are densely infiltrated with polymorphonuclear leukocytes. (Negative 71041.)

base and walls of the ulcer. At the same time, blood vessels grow into the cornea to supply antibodies and to initiate the processes of repair, which proceed in the same fashion as in the healing of wounds elsewhere. The mere fact that an infected corneal ulcer has perforated is not the catastrophe that it first appears to be. The loss of aqueous induces the formation of a plasmoid aqueous rich in antibodies, and as this washes over the infected area it frequently speeds up the healing process. However, perforation can lead to the introduction of the infection into the interior of the eye or to incarceration of ocular tissue in the wound.

At times a virulent organism, such as the pneumococcus, attacks the cornea of a person with diminished resistance, and the spread of the toxin produces a marked response. The diffusion of this toxin to the pericorneal vessels induces a dense infiltration of pus cells into the cornea around the ulcerated area. Also, its diffusion through the intact Descemet's membrane has a marked chemotactic influence on the con-

tents of the vessels of the iris and the ciliary body. There is an extensive migration of white blood cells, chiefly leukocytes, from the dilated blood vessels into the anterior chamber. This is accompanied with fluid, which

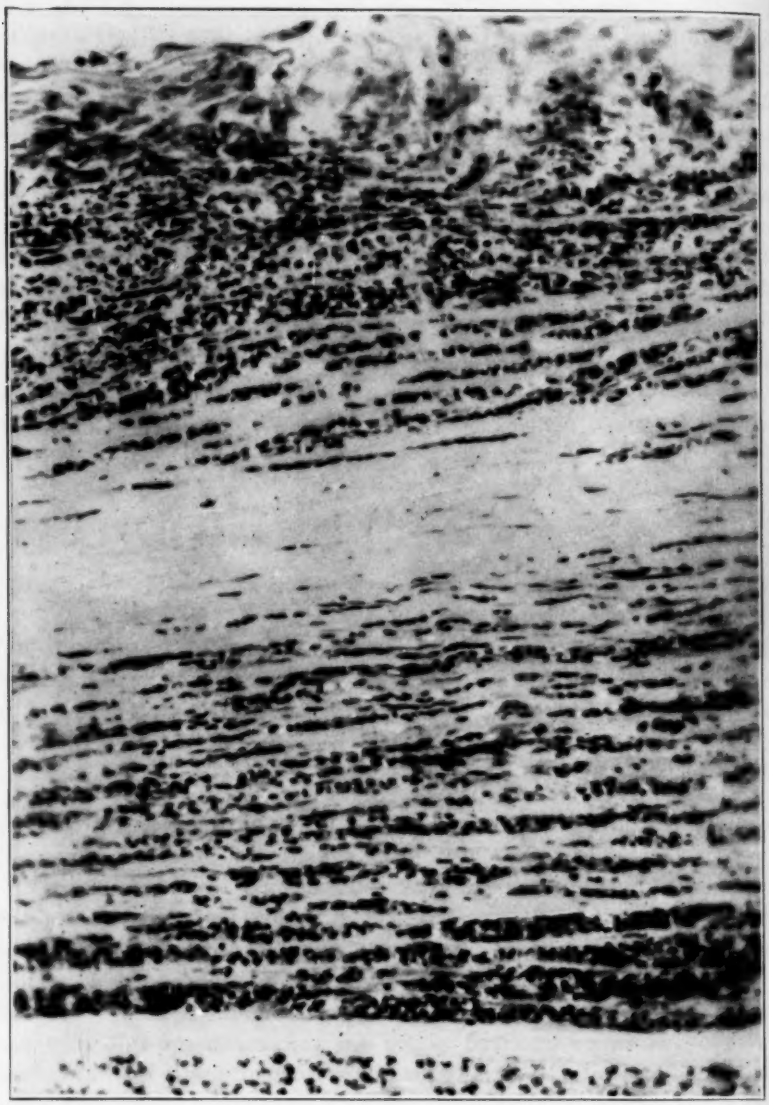


Fig. 9.—Purulent keratitis. There is ulceration of the superficial layers. The stroma is densely infiltrated with polymorphonuclear leukocytes. A purulent exudate is present in the anterior chamber. (Negative 71036.)

later has an increased fibrinous content and becomes semisolid. The resulting hypopyon is sterile, since the organisms cannot pass through an

intact Descemet's membrane. With the healing of the corneal lesion there is a gradual resorption of the exudation in the anterior chamber. Occasionally there is seen associated with the hypopyon ulcer an infiltration of purulent cells into the posterior stroma. This type of abscess may lie between the stroma and Descemet's membrane or even between the layers of the fragmented Descemet's membrane. It is difficult to judge whether such posterior corneal abscesses result from leukocytes migrating from the peripheral vessels and attacking Descemet's membrane from the front or whether they are derived from the hypopyon and destroy Descemet's membrane from behind.

A type of corneal inflammation usually caused by the pneumococcus is clinically termed ulcus serpens and is particularly severe. The organisms penetrate through the abraded epithelium and grow between the lamellas, causing extensive necrosis. Dense masses of leukocytes infil-

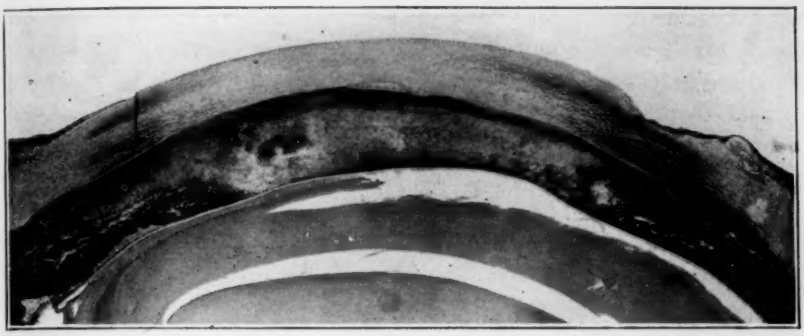


Fig. 10.—Ring abscess of the cornea. The epithelium is desquamated. The stroma is densely infiltrated at the periphery by partially necrotic polymorphonuclear leukocytes. Central infiltration is not marked. Purulent exudate fills the anterior chamber. (Negative 71035.)

trate the cornea, particularly near the site of greatest bacterial growth. Healing processes are initiated on one side, while the opposite side of the ulcer creeps ahead. The infiltration may involve the deeper layers of the cornea, forming a posterior abscess, and may lead to hypopyon and iridocyclitis. Perforation may also occur, with loss of the eye from invasion of bacteria.

Another severe form of corneal inflammation is the ring abscess, which manifests itself clinically as a rapidly developing zone of infiltration separated from the periphery by a clear zone. The entire central portion of the cornea becomes necrotic, and panophthalmitis usually ensues. Histologic examination shows a massive leukocytic infiltration in the peripheral zones, not extending to the limbus. The heaviest infiltration is in the deeper corneal layers. The whole central region is necrotic, and the leukocytic ring represents the defensive attempt to wall off and remove this necrotic area.

NONPURULENT KERATITIS

Nonpurulent keratitis is an infiltrative inflammation of the substantia propria, clinically termed parenchymatous or interstitial keratitis. Etiologically it is most frequently associated with syphilis, tuberculosis, focal infections and leprosy, although it is often allergic in origin.

The interstitial keratitis associated with congenital syphilis is often accompanied with anterior uveitis. Spirochetes have been found in the deeper corneal layers but are sparse. Owing to the extensive necrosis and the scarcity of organisms, an allergic mechanism has been frequently suggested as the cause of this inflammation. Pathologically the disease is essentially edema and necrosis of the corneal lamellas producing massive infiltration of wandering cells, chiefly lymphocytes. Blood vessels grow in toward the lesion in a characteristic palisade. These changes occur most markedly in the periphery, where the deeper corneal layers become heavily infiltrated and vascularized. In cases of especially severe involvement nodular aggregations of lymphocytes form miliary gummas. Healing occurs partly with the proliferation of the fixed corneal corpuscles and partly with fibrous tissue derived from the invading blood vessels. These vessels run at different levels, depending on the depth of the inflammatory process. Several systems of blood vessels may be observed, with comparatively few anastomoses between the various levels. Although the substantia propria bears the brunt of the inflammation, Bowman's and Descemet's membranes and also the endothelium become involved, while the surface epithelium usually escapes. With the progression of the reparative processes, the blood vessels extend farther into the cornea and the necrotic tissue is removed, being replaced by scar tissue. The scar tissue and the blood vessels constitute permanent changes in the stroma, remaining even after the active inflammation has completely subsided.

One form of tuberculous interstitial keratitis shows many of the characteristics of the syphilitic type and has been assumed to be an allergic reaction to the tuberculoproteins. There is another, more common form, in which infiltrates spread in the deeper corneal layers from lesions in the sclera at the limbus. Clinically these corneal opacities are accompanied with mutton fat deposits on the posterior corneal surface and by nodules in the iris. Tubercle bacilli are very rarely found, although foci of tuberculous granulation tissue are present around Schlemm's canal. At times the entire cornea may become a caseous mass.

There are other clinical types of parenchymatous keratitis, which have been called keratitis profunda, disciform keratitis, etc. The former

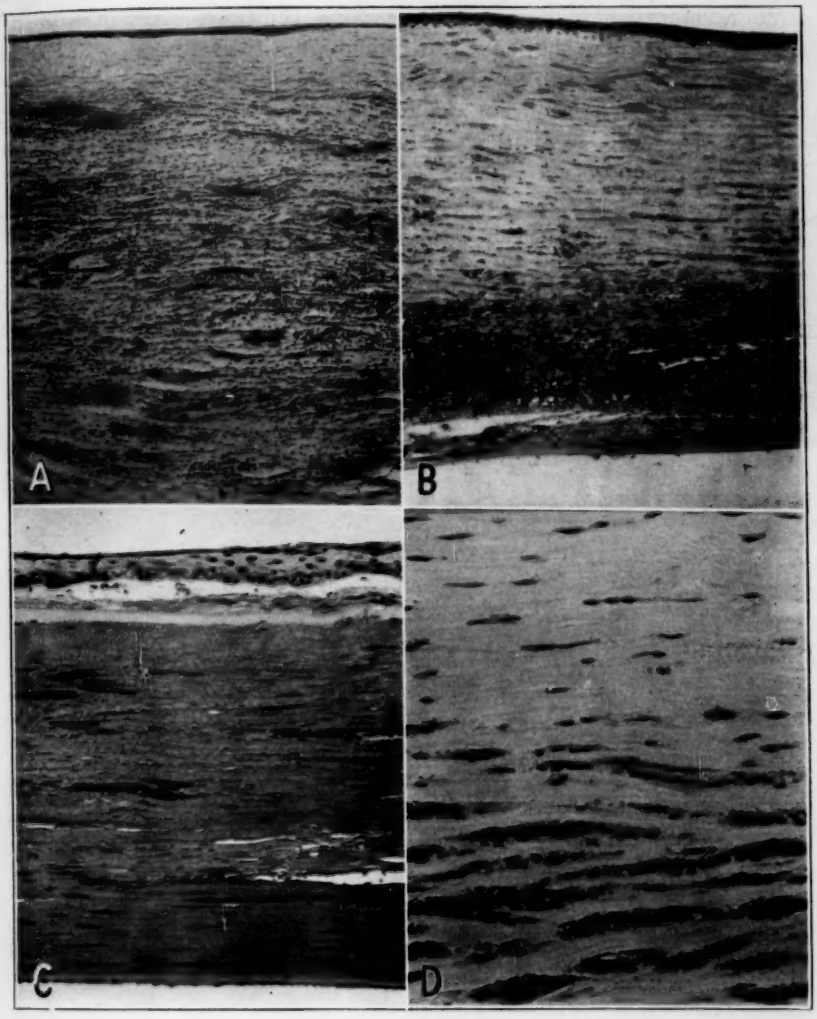


Fig. 11.—*A*, nonpurulent keratitis. The stroma is densely infiltrated with lymphocytes and plasma cells. (Negative 71059). *B*, keratitis profunda associated with granulomatous scleritis in a case of tuberculous kidney. There are round cell infiltration and vascularization in the deeper layers of the stroma. Around one of the emissary veins of the sclera a tubercle was found, with giant and epithelioid cells. No tubercle bacilli were seen. The Wassermann reaction was negative. (Negative 71058.) *C*, interstitial keratitis in a case of hereditary syphilis. The cornea is vascularized throughout, most of the vessels being in the middle layers. The stroma shows formation of scar tissue. There is moderate lymphocytic infiltration in the central portion of the cornea. The inflammation is subsiding. (Negative 71052.) *D*, dense infiltration of lymphocytes widely separating the lamellas. (Negative 71056.)

apparently is a less chronic condition than those already discussed and is not accompanied by deep vascularization. Infiltrates occur deep in the substantia propria in the central zones and are usually absorbed, although at times they leave permanent scars. The condition is regarded as due to an allergic reaction to some chronic focus of infection. A few cases of disciform keratitis have been studied pathologically, and the essential change was found to be massive necrosis of the lamellas with comparatively slight lymphocytic infiltration. Later vascularization occurs, leaving permanent scars, and, as this is often associated with herpes, it has been suggested that the cause is neurotrophic.

Since the cornea is normally avascular, it combats the pathologic processes of inflammation by the invasion of newly formed blood vessels.

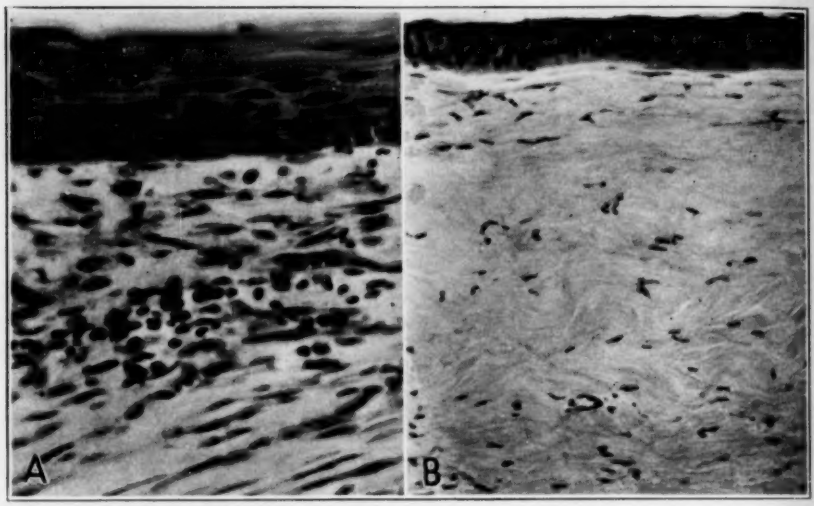


Fig. 12.—*A,* chronic nonpurulent keratitis. The stroma is infiltrated with polymorphonuclear leukocytes and lymphocytes. Bowman's membrane is fragmented. (Negative 71037.) *B,* old keratitis with hyaline and vascular scarring. Scanty infiltration of lymphocytes and polymorphonuclear leukocytes may be noted. (Negative 71039.)

Julianelle and Lamb [14] have demonstrated that in superficial inflammations of the stroma capillary branches bud off from the adjoining conjunctival vessels and run their course between the lamellas toward the foci of necrosis. This superficial vascularization is present in cases of phlyctenular keratitis, acne rosacea and trachoma and particularly in the vascularization of an ulcer. In the deeper forms of corneal inflammation the new vessels are derived from the deep ciliary vessels and run a

14. Julianelle, L. A., and Lamb, H. D.: Studies on Vascularization of the Cornea, Am. J. Ophth. **17**:916, 1934.

straight, nonanastomosing course, since they are constrained by the corneal lamellas. However, when necrosis of the lamellas occurs, the invading vessels cross from one plane to another, and anastomoses between different systems may occur. Julianelle and Bishop [15] studied

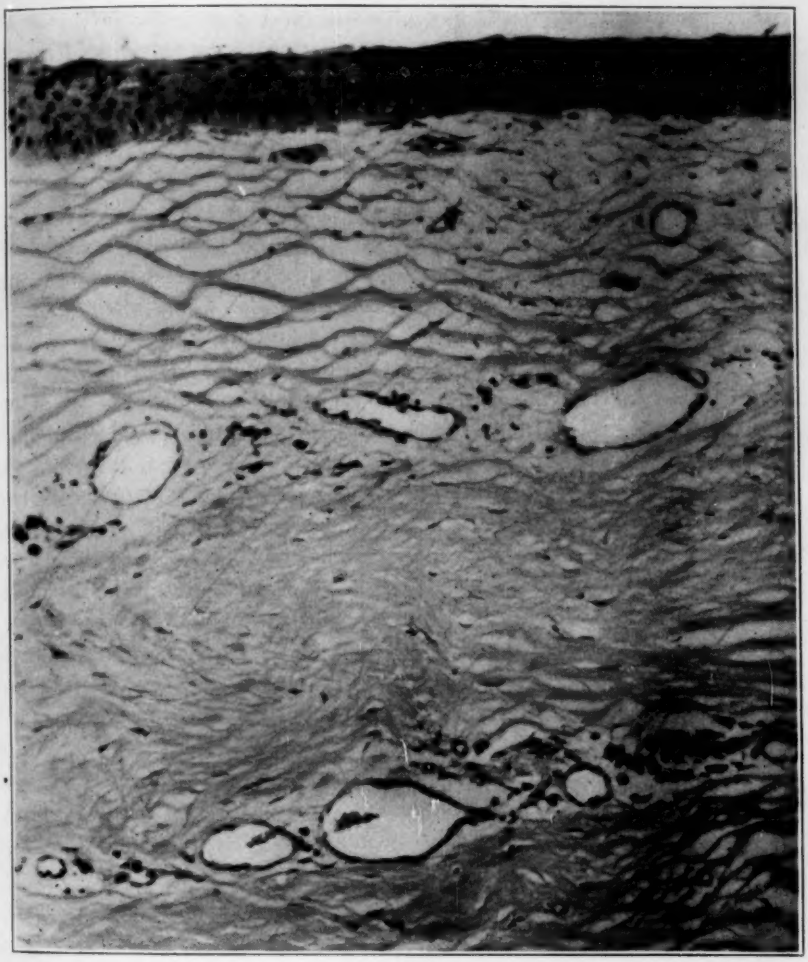


Fig. 13.—Old keratitis with extensive vascularization and perivascular infiltration, predominantly by plasma cells. (Negative 71042.)

the development of blood vessels in the sensitized cornea. They found that the injection of a specific antigen into the sensitized cornea induced an inflammatory response accompanied by the development of new

15. Julianelle, L. A., and Bishop, G. H.: The Formation and Development of Blood Vessels in the Sensitized Cornea, Am. J. Anat. **58**:109, 1936.

vessels. These originated below the sclerocorneal margin and were initiated by petechial hemorrhages, which were rapidly penetrated by capillaries springing from the conjunctival vessels and growing into the lamellar interspaces in the form of loops. In the foregoing section on the repair of wounds it was mentioned that the sequence of events is partly the regeneration of corneal tissue and partly the laying down of fibrous tissue derived from the granulation tissue introduced by the newly formed blood vessels. The same processes occur in the repair of inflammation in the corneal stroma.

DYSTROPHIES

The familial corneal dystrophies, comprising the nodular type of Groenouw and the reticular type of Haab, have shown considerable pathologic discrepancy. The essential feature is the deposition of hyalin between the lamellas of the superficial stroma. There then occurs a swelling and disintegration of the lamellas and the corpuscles.

The pathologic picture of Salzmann's nodular dystrophy is also diverse. It differs from the familial type in that no hyaline or amorphous material is deposited. Brown and Katz [16] studied excised nodules in a case of this condition and found that the superficial layers of the stroma are the first involved. The cells show evidence of pyknosis, and the intercellular connective tissue is condensed into an almost homogeneous mass. Later the stroma shows lamellar fibrillation. As the deeper layers become involved, the superficial layers show more marked changes, with some cellular infiltration and vascularization.

Keratoconus is considered by some to be a dystrophy of the cornea. No pathologic changes are present until late in the condition, when ruptures in Descemet's membrane occur, allowing aqueous to filter into the stroma and cause opacities at the apex. There are no evidences of inflammation. Mutch and Richards [17] were able to produce keratoconus experimentally in rats by putting them on a diet free from vitamin A. In most cases the cornea regained its normal contour after a few weeks' dosing with vitamin A, although corneal nebulae remained as permanent defects.

Lieut. Col. J. E. Ash and Mrs. Helenor C. Wilder, of the Army Medical Museum, assisted in the preparation of this material.

16. Brown, E. V. L., and Katz, D.: Salzmann's Nodular Corneal Dystrophy, Arch. Ophth. **13**:598 (April) 1935.

17. Mutch, J. R., and Richards, M. R.: Keratoconus Experimentally Produced in the Rat by Vitamin A Deficiency, Brit. J. Ophth. **23**:381, 1939.

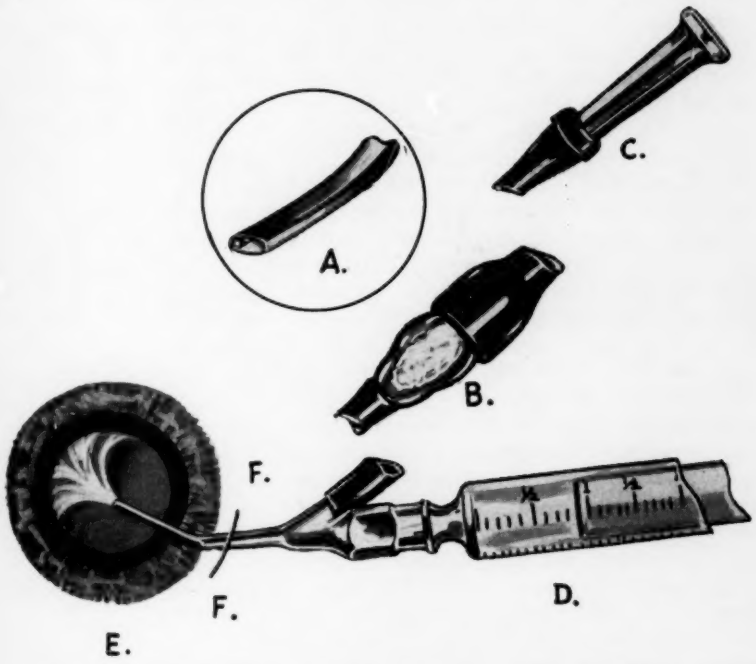
Clinical Notes

REMOVAL OF SOFT CATARACT BY SUCTION

A New Double-Barreled Aspirating Needle

OTIS WOLFE I, M.D., AND R. M. WOLFE, M.D.,
MARSHALLTOWN, IOWA

We present a new combined irrigating and suction tip which greatly simplifies the technic for extraction of soft cataract. It consists of a double-barreled needle for injecting solution into the anterior chamber while oral suction is maintained through the second, or larger, barrel.



The double-barreled suction tip (*A*) is inserted into the anterior chamber (*E*) through the keratome incision (*FF*). The contents of the lens are drawn up into the glass trap (*B*) by oral suction (*C*). Physiologic solution of sodium chloride is simultaneously injected into the anterior chamber by means of the syringe (*D*).

A narrow keratome picks up the conjunctiva 3 mm. back of the limbus and enters the anterior chamber. The anterior capsule is slit in the ninetieth meridian by the point of the keratome. The double-barreled needle is inserted and the lens cortex aspirated. Fluid is then

injected and the aspirating continued until the Hildreth lamp shows little or no lens cortex remaining.

Sometimes a preliminary discission of the anterior capsule is made and the aqueous allowed to soften the lens for two or more days before aspiration is applied. This depends on just how soft the lens is estimated to be when examined by the slit lamp. If the posterior capsule is opaque it is slit as the final step of the technic. In most cases, particularly in those of perinuclear cataract, discission is not necessary, although in about 25 per cent it may be required later. The technic is also ideal for removing the lens in cases of high myopia and toxic cataract (dinitrophenol cataract, etc.). The operative reaction is slight, and refraction can be done after a few days. Little scar or astigmatism results, and contact lenses can be fitted easily.

After seventeen years of experience in well over 200 cases, we believe that this method, whenever applicable, closely approaches the ideal.

Correspondence

THIAMINE (VITAMIN B_1) IN OPHTHALMOLOGY

To the Editor:—I was very much interested in the article entitled "Vitamin B in Ophthalmology" by C. V. Veasey, which appeared in a recent number of the ARCHIVES (**25:** 450 [March] 1941). The author summarized the literature on this complex subject, in addition to reporting the results of his own work. It seemed to me, however, that several points warrant further comment.

It is perhaps of academic interest only, but the statement that thiamine (or vitamin B_1) acts as a catalyst in the combustion of carbohydrate is not in accord with present knowledge, since, as Lohmann and Schuster (*Naturwissenschaften.* **25:** 26, 1937) have pointed out, it is not thiamine itself but a salt (thiamine pyrophosphate; cocarboxylase) which is the catalyst for the decarboxylation of pyruvic acid.

Veasey properly reiterated the claims that lack of vitamin B_4 "prevents a specific paralysis in rats and is necessary for chicks" but he did not call attention to the fact that Raeder (*Biochem. J.* **23:** 689, 1929), who originally thought she had discovered a new vitamin (B_4) (Kinnersly, H. W.; O'Brien, J. R., and Peters, R. A.: *Biochem. J.* **29:** 701, 1938), has since found that the observed symptoms could be relieved with thiamine. It would seem that it is extremely doubtful whether there is any such factor as vitamin B_4.

Of particular importance, however, is the reference to the work of F. P. Fischer (*Arch. d'opht.* **2:** 108, 1938). Veasey reported Fischer's finding of what would appear to be an incredibly small amount of thiamine in the lens, viz., 0.001 microgram. For the detection of thiamine Fischer employed Jansen's method (*Rec. d. trav. chim. d. Pays-Bas* **55:** 1046, 1936), by which this substance can be oxidized to thiochrome; the latter is then detected fluoroscopically. Jansen could detect as little as 1 microgram of pure thiamine and was able to recover "nearly all" of 5 micrograms of thiamine which he had previously added to yeast. It is not clear how, with no appreciable change of method, Fischer could detect, amid many other fluorescing compounds in the lens, a quantity of thiamine one thousandth the minimum Jansen reported for pure solutions. Fischer gave no details as to what kind of apparatus was used, how many lenses were involved, what blank was subtracted, if any, for interfering substances or what filters were used to isolate the fluorescent radiations, to mention but a few of the more obvious omissions. Nor did he explain why he did not follow the usual procedure, as did Jansen, of adding some pure thiochrome to the lens material to see whether or not his method was adequate to detect amounts of thiochrome corresponding to 0.001 micrograms of thiamine.

On the evidence reported, it is impossible to ascertain why Fischer assumed that the fluorescence, if any, was caused by thiochrome. That such an assumption was apparently unjustified is suggested by Fischer himself in a later paper (*Ophthalmologica* **96:** 219, 1938), in which he claimed that the substance present in the lens is really 0.0012 micro-

grams of thiamine pyrophosphate. This quantity, he stated, corresponds exactly to 0.001 micrograms of thiamine per lens as reported previously. While in the second paper he mentioned the number of lenses used and described how they were prepared, he again gave no details concerning the analytic procedure. Concerning the significance of this 0.0012 micrograms of cocarboxylase, Fischer stated that the results were in good agreement with those of Goudsmit and Westerbrink (*Nederl. tijdschr. v. geneesk.* **81:** 2632, 1937; *Acta brev. Neerland.* **8:** 168, 1938), who found 7 to 13 micrograms of cocarboxylase in the liver, and that "the values for the lens are not so extremely small, if one must take into account the difference in weight between the lens and liver." Fischer would have been less misleading had he mentioned that his comparison of the weight of the lens and liver and their relative cocarboxylase content was based on the lens of an ox (2.5 Gm.) and the liver of a rat (10 Gm.)!

It is my object not to affirm or to deny the existence of thiamine or of its diphosphate in the lens but to point out that the evidence for their existence, particularly in functional amounts, is questionable to say the least and that thus the therapeutic implications (Fischer) drawn from its alleged absence in cataract are without foundation.

Undoubtedly it was not Veasey's primary object to appraise each paper he reviewed, but it seems to me that repeated uncritical citation of work frequently leads to the highly unsatisfactory situation of lending unmerited credence to a certain research.

V. Everett Kinsey, Ph.D., Boston.

News and Notes

EDITED BY W. L. BENEDICT, M.D.

GENERAL NEWS

Brazilian Congress of Ophthalmology.—Dr. Harry S. Gradle, Chicago, president of the Pan-American Congress of Ophthalmology, was a guest at the fourth Brazilian Congress of Ophthalmology, held in Rio de Janeiro June 26 to July 1. Dr. Gradle delivered an address on "Glaucoma Capsulare." Prof. Octavio Rego Lopes, Rio de Janeiro, was president of the congress, and Dr. Natalicio de Farias, Rio de Janeiro, was secretary. Members of the committee in charge of the scientific program included Drs. Ivo Corrêa Meyer and Santayana Mascarenhas, Porto Alegre; Linneu Silva, Minas Geraes; Moacyr E. Alvaro, Souza Martins and J. J. Britto, São Paulo; Isaac Salazar, Recife; Cesario de Andrade and Francisco Ferreira, Bahia; Paulo Cesar Pimentel, Rio de Janeiro; Deusdedith Coelho Duarte, Pará, and Leonidas Ferreira, Paraná. Official subjects of discussion chosen for the congress were the relation between ophthalmology and legal medicine, the constitution in ophthalmology and the vision of school children.

The program was as follows:

June 26 (Thursday)—Preparatory session at 4 p. m.; solemn session of opening of the congress at 9 p. m.

June 27 (Friday)—Visit to the Oswaldo Cruz Institute, the Medico-Pedagogical Centers and the departments of ophthalmology of the Central Army Hospital; session of social ophthalmology at 2:30 p. m. and that of clinical ophthalmology at 8:30 p. m.

June 28 (Saturday)—Visit to the Institute of Public Education and the Department of Organization of Hospitalas of the General Secretariat of Public Health of the Prefecture of the General Districts; session of social ophthalmology at 2:30 p. m. and that of clinical ophthalmology at 8:30 p. m.

June 29 (Sunday)—Visit to the city and municipality of Rio de Janeiro, the Correas Sanatorium and the Emperador Museum; lunch at the Tennis Club.

June 30 (Monday)—Visit to Guanabara Bay and Viana Island; session of social ophthalmology and clinic at 9 p. m.

July 1 (Tuesday)—Session at 8:30 a. m. for presentation of the conclusions and resolutions of the congress, organization of a central office and determination of the regulations and date for the next congress; lunch at 12 noon in Gavea Park; reception by the President of Brazil in the afternoon; closing session in the evening.

The articles read during the congress had to be sent before June 10 to Dr. Natalicio de Farías. Each speaker was allotted ten minutes. When the reading of all the articles was over, every congressionist could speak once, for five minutes.

Home Study Courses for 1941-1942.—The American Academy of Ophthalmology and Otolaryngology will again offer its home study courses in the fundamentals of the two specialties, beginning August 1.

These courses were intended primarily for residents in specialized institutions that give no formalized instruction in fundamentals. The committee reports, however, that many physicians preparing for examinations given by special boards and many who were practicing in small communities without facilities for postgraduate instruction were included in the first year's registration of 465. A registration of about 150 had been expected.

Each registrant received on the first of each month a list of reading in standard textbooks designated by chapters. At the end of the month twenty questions covering this material was sent to the registrant. These were to be answered and returned to the committee. For each month one physician was in charge of correcting and grading the papers, aided by a group of assistants, all diplomates of the special boards. Corrected papers were returned to the registrant.

The following topics were covered last year:

	Ophthalmology	Otolaryngology
August	Anatomy, histology, embryology	Anatomy I
September	Optics and visual physiology	Anatomy II
October	Physiology	Histology-pathology
November	Pathology	Physiology
December	Refraction	Bacteriology
January	Refraction	Biochemistry
February	Perimetry	Otolaryngologic examination
March	Muscles	Hearing tests
April	Biomicroscopy and ophthalmoscopy	Vestibular tests

The cost of the course is $10. The work is started August 1 in order that residents entering their service July 1 may have a month for orientation. The committee emphasizes that the course is for training in fundamentals and in no way equips a physician for clinical practice. Registration should be in the hands of Dr. William P. Wherry, 1500 Medical Arts Building, Omaha, by July 15.

SOCIETY NEWS

Virginia Society of Oto-Laryngology and Ophthalmology.—The twenty-second annual meeting of the Virginia Society of Oto-Laryngology and Ophthalmology was held in Richmond on May 10, 1941, under the presidency of Dr. G. G. Hankins, Newport News, Va. Dr. James Babbitt and Edmund B. Spaeth, Philadelphia, were guest speakers, and their subjects were "Geriatrics and Its Role in Otology" and "Surgical Treatment of Neurofibroma of the Orbit," respectively. The following officers were elected: president, Dr. Mortimer H. Williams, Roanoke, Va.; president-elect, Dr. Guy Fisher, Staunton, Va., and secretary-treasurer, Dr. Meade Edmunds, Petersburg, Va. The next meeting will be held in Staunton in May 1942.

North Dakota Academy of Ophthalmology and Oto-Laryngology.—Dr. Albert E. Spear, Dickinson, N. D., was elected president of the North Dakota Academy of Ophthalmology and Oto-Laryngology at the annual meeting in Grand Forks, N. D., on May 20, 1941. Dr. Le Roy G. Smith, Mandan, N. D., was elected vice president and Dr. Frederick L. Wicks, Valley City, N. D., secretary. The guest speaker was Dr. Kinsey M. Simonton, Rochester, Minn., and his subject was "The Symptom of Dizziness: Its Significance in General Practice."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

THE SIGNIFICANCE OF THE SPECIFIC INTRADERMAL REACTION, OF THE ERYTHROCYTE SEDIMENTATION RATE AND OF THE DIFFERENTIAL WHITE CELL COUNT IN CASES OF EXPERIMENTAL OCULAR TUBERCULOSIS. K. HISHI, Arch. f. Ophth. **141**:633, 1940.

The object of the study described was to determine whether a purely ocular experimental tuberculous disease produces any systemic effects. Healthy adult rabbits were infected by injection of an accurately measured amount of an emulsion of live tubercle bacilli of the bovine type into the anterior chamber or into the vitreous. The infected eyes showed ocular tuberculosis of varying severity and were enucleated from twenty-four to twenty-nine days after the inoculation. Postmortem examination two to four months after the inoculation revealed hematogenic metastases in only 1 of 10 animals. In search of systemic effects of the ocular disease, the following observations were made once a week during the two months following the inoculation: the differential white blood cell count, the erythrocyte sedimentation rate and the reaction to the Mantoux test (intradermal injection of a 1 per cent solution of old tuberculin). During the acute phase of the ocular tuberculosis the white blood cell count showed, in addition to moderate absolute leukocytosis, a relative increase of the neutrophilic polymorphonuclear leukocytes with relatively few multilobulated cells, and the erythrocytic sedimentation rate was greater than normal. After enucleation of the infected eye both the white cell count and the sedimentation rate returned to normal fairly promptly. The Mantoux reaction became positive during the second week after the inoculation and remained positive despite removal of the tuberculous focus.

The author takes issue with Loewenstein's theory that a tuberculous disease of an organ or a tissue causes lowered resistance to reinfection of a homologous organ. Eight rabbits in which the infected eye was not enucleated had extensive hematogenic metastases. Only 2 of these animals showed metastasis to the other eye; this took the course of a chronic caseating tuberculoma of the choroid and not that of rapidly progressive exudative uveitis as in the originally infected eye. These observations did not suggest unusual sensitivity to reinfection of the second eye. Experimental superinfection of the second eye also seemed to produce a less severe disease than that of the eye originally infected. Thus it does not appear that a tuberculous disease of one eye predisposes its mate to tuberculous infection.

P. C. KRONFELD.

Conjunctiva

IRRIGATIONS WITH SULFANILAMIDE AS A TREATMENT FOR GONORRHEAL CONJUNCTIVITIS. W. J. REIN and O. B. TIBBETTS, Am. J. Ophth. **22:** 1127, 1939.

Rein and Tibbetts examine all patients with conjunctivitis bacteriologically, and if gram-negative intracellular diplococci are found the patient is classed as having gonorrheal ophthalmia and is admitted to the hospital. The eye is irrigated every fifteen minutes night and day with a 0.5 per cent solution of sulfanilamide made up in physiologic solution of sodium chloride. If other complications exist (vaginitis or urethritis), the patients are given 30 to 60 grains (1.8 to 3.6 Gm.) of sulfanilamide orally. The following summary is given:

"1. A series of 15 cases of gonorrheal ophthalmia have been reported. We believe a new technique in the local use of sulfanilamide has been advanced.

"2. We feel that the local use of sulfanilamide in solution in the treatment of gonorrheal ophthalmia practically eliminates the danger of systemic reactions.

"3. In our series of 15 cases of gonorrheal ophthalmia treated locally with sulfanilamide there have been no ocular complications.

"4. The economic advantages, by reason of the reduction of the period of hospitalization to less than one third that required by use of other standard measures, are obvious."

W. S. REESE.

Cornea and Sclera

SPECIFIC INFLAMMATION OF THE CORNEA IN CHICKENPOX. A. LOWENSTEIN, Brit. J. Ophth. **24:** 391 (Aug.) 1940.

The author points out that vitiligo iridis has been observed after smallpox, experimentally from injected virus of herpes febrilis (herpes iridis usually occurring with relapsing hemorrhages into the anterior chamber) and after chickenpox. A case is reported in which specific keratitis and iritis occurred in the course of chickenpox. The following summary is supplied by the author:

"The previously unknown clinical picture of corneal infection with chickenpox virus is described, with slit-lamp pictures. An earlier observation of the author, who had found vitiligo iridis after chickenpox, is thus completed. The new recognised forms of vitiligo iridis in smallpox, chickenpox, herpes iridis (zoster), inoculation iritis in rabbits (herpes febrilis), and scarlet fever present a unity from which we can deduce a biological relationship between their germs."

The article is illustrated.

W. ZENTMAYER.

DISK-SHAPED DEPOSIT OF CRYSTALS IN THE CENTER OF THE CORNEA: A HEREDITARY DISEASE; REPORT OF CASES. W. F. SCHNYDER, Klin. Monatsbl. f. Augenh. **103:** 494 (Oct.-Nov.) 1939.

The author describes 4 cases of disk-shaped deposits of crystals in the center of the cornea and renders an anamnestic account of 2 other

members of the same family afflicted similarly. The deposits began in early childhood, increased but slightly during life and never caused any inflammatory or other irritations. The deposits were located in the outer parenchyma, directly beneath Bowman's membrane. In every case a white "limbus belt" (type I of Vogt) was noted at the nasal and temporal limbus, within the area of the palpebral fissure. The disease, described first in 1927, represents a hitherto unknown type of corneal degeneration which is inherited dominantly.

K. L. STOLL.

TWO BROTHERS WITH KERATOCONUS. M. KAELIN-SULZER, Klin. Monatsbl. f. Augenh. **103:** 513 (Oct.-Nov.) 1939.

Two brothers, aged 30 and 26 respectively, showed keratoconus of the same appearance, with similar central opacities of the same form and extent. No other members of the family were known to have or to have had similar ocular symptoms. Astigmatism of a high degree was present in the mother's eyes. Consanguinity could not be proved, nor could it be entirely excluded, because the parents of the mother had the same family name. The relation of the mother's parents may have gone farther back than could be demonstrated at the time of observation.

K. L. STOLL.

Experimental Pathology

REGENERATION OF THE CORNEAL NERVES AFTER LINEAR EXTRACTION. I. EGOROV, Vestnik oftal. **16:** 3, 1940.

The experiment was done on 32 guinea pigs of the same age and weight. The incision of the cornea (61 eyes) was done with the animal under ether anesthesia; the postoperative course was uneventful, and the time of observation was ninety-six days. The corneal specimen was dyed with methylene blue (methylthionine chloride), by which the condition of the nerve elements and the morphologic changes in Schwann's nuclei are well and simply demonstrated.

The cornea was opaque during the first few days after the operation, but the opacities cleared in about two weeks, when union of the lips of the wound was complete. The newly formed connective tissue of the scar was richly vascularized; the loops of the vessels were located in the superficial and deep layers of the substantia propria. The process of regeneration of the corneal ciliary nerves is described in detail, and eight photomicrographs illustrate well the consecutive changes at various times after operation, from the twelfth to the eighthieth day.

The conclusions are as follows: The use of methylene blue is a good simple method for following up the regeneration of the innervation apparatus of the operatively treated cornea. In typical cases the regeneration of the nerve elements in the corneal scar was complete at the end of the third month. This method also allows simultaneous study of the reactive growth of the nerve fibers at a distant location from the site of the trauma. It was frequently observed that the severed nerve fibers regenerated not only at the end but in distant places; collateral bundles of regeneratively growing fibers made their way beside

the connective tissue and penetrated and grew into the stroma of the scar. The reaction of the nerve elements was also demonstrated by the structural shifting of distant sectors by proliferation of small branches which had dilated ends. Hypertrophy of the "end" lamellas was also noted.

It is most probable that the regenerative proliferation of the nerve is in close relation with the innervated tissues and with the ensuing changes, as at the final phase of differentiation of the scar tissue the previously increased number of nerve elements became normal again.

O. SITCHEVSKA.

General Pathology

STUDIES ON SURFACE-EPITHELIUM INVASION OF THE ANTERIOR SEGMENT OF THE EYE. T. L. TERRY, J. F. CHISHOLM JR. and A. L. SCHONBERG, Am. J. Ophth. **22**: 1083 (Oct.) 1939.

Twenty-eight cases of epithelization of the anterior or the posterior chamber are reported. The authors draw the following conclusions:

"1. Accidental perforation is more prone to be complicated by epithelization than operative perforation. The presence of prolapsed material favors extension of epithelium into the eye by proliferation. In adult life, epithelization is more frequent in males than in females.

"2. Endothelium on the back of the cornea is a barrier to the growth of epithelization. However, structures derived from neural ectoderm (iris pigment epithelium, ciliary epithelium and retina) are not barriers.

"3. The gray line seen clinically, marking the delineation of growth, is interpreted as a piling-up of epithelium at the growing age.

"4. Glaucoma does not invariably result from epithelization. When glaucoma is produced, the epithelium grows over the iris-angle meshwork or may block it by cells exfoliated into the aqueous."

W. S. REESE.

Injuries

EXPERIMENTS ON THE EFFECT OF ASCORBIC ACID IN MUSTARD GAS BURNS OF THE EYE. I. MANN and B. D. PULLINGER, Brit. J. Ophth. **24**: 444 (Sept.) 1940.

The present research has been done in response to an invitation from Livingston and Walker to determine whether their suggestion that ascorbic acid intravenously or parenterally may prevent or counteract the peculiar chemical and physical effects of mustard gas (dichloroethyl sulfide) is correct. The authors reach the following conclusions:

1. Intravenous injections of ascorbic acid do not prevent or influence the progress of mustard gas lesions of the lids, the conjunctivas or the corneas.

2. The degree of damage is related to the dose and is not influenced by resistance of individual animals to the chemical. Identical doses produce almost identical lesions.

W. ZENTMAYER.

TREATMENT BY ALCOHOL OF CORNEAL INJURIES BY INDELIBLE PENCIL: CLINICAL AND EXPERIMENTAL STUDY. JEAN SEDAN, Ann. d'ocul. **177:** 65 (Feb.) 1940.

The author in 1922 observed an extremely serious wound of the cornea in a student who had pierced the left eye with the point of an indelible pencil. Despite early and energetic treatment, a true interstitial keratitis, bluish black, developed, causing functional loss of the eye, which for about ten days was in great danger. The hypopyon which was present showed absolute indifference to medical and surgical treatment. In 1938 he had occasion to study a similar case, in which, despite the extreme rapidity of the appearance of the keratitis, a practically complete anatomic and functional cure was obtained.

Similar treatment was tried on animals to see whether the therapeusis so successful for 1 patient could be recommended. He gives the details of the animal experimentation. Some of the eyes were treated by washing with 10 per cent tannic acid; others were washed and decolorized with oxygenated water, and others were cleaned, decolored and washed with 90 per cent alcohol. In the 7 eyes treated with alcohol more or less useful vision was obtained; treatment with tannic acid oxygenated water was unsuccessful. A bibliography and one illustration are presented.

S. H. MCKEE.

Lacrimal Apparatus

INDIRECT STRICTURES OF THE LACRIMAL CANALICULI. A. FAZAKAS, Arch. f. Ophth. **141:** 662, 1940.

The author distinguishes between direct strictures of the canaliculi due to inflammatory swelling of their walls and indirect, or cicatricial, strictures. Direct strictures are promptly removed by instillation of epinephrine hydrochloride or by one irrigation of the lacrimal passages. Cicatricial strictures may be the result of local fungous infections, of severe forms of conjunctivitis or of a hordeolum situated near a canaliculus. The shape and extent of the indirect strictures vary. The treatment consists of gentle probing with conical probes. Of 319 indirect strictures, 235 were cured after one probing; 65 were cured after two probings, and 19 were cured after three or more probings.

P. C. KRONFELD.

Lens

A CASE OF EXFOLIATION OF THE LENS CAPSULE WITH PATHOLOGICAL REPORT. A. GARROW and I. C. MICHAELSON, Brit. J. Ophth. **24:** 400 (Aug.) 1940.

The patient was a man aged 69. The right eye was removed for sarcoma of the choroid. For examination of the eye the pupil was dilated, and the slit lamp revealed well marked exfoliation of the lens capsule. There were a central film with curled frayed edges and a coarse-grained peripheral film also curved over in parts. No exfoliated

particles were seen adherent to the pupillary edge of the iris or elsewhere. The left eye also showed exfoliation of the lens capsule.

Pathologic examination of the capsule showed some peeling of its most superficial layers. This was irregular in distribution and was present only in the anterior capsule. The tufted appearance which resulted consisted of long thin strands of tissue and of short pointed projections representing the crests of undulations on the surface of the lens capsule. (These changes were shown in photomicrographs.) At no place could the marked thinning of the capsule or the feltwork appearance of the tissue described by Vogt be made out. The subcapsular epithelium appeared normal, but in one place it was duplicated. No debris of detached capsule could be recognized in the angle of the anterior chamber. Vogt described 5 cases in which he had the opportunity of making an anatomic study. In these he described the separation of the anterior fibers as being somewhat similar to the normal passage of the zonule from the lens capsule, except that it was more axial and in the opposite direction. Open feltwork appearance, peeling and degeneration were the apparently progressive changes noted by him.

The changes described and illustrated in this paper are essentially the same as those recorded by Vogt, and the case presented is of some interest as the first to be described pathologically from England.

The article is illustrated.

W. ZENTMAYER.

Methods of Examination

A NEW METHOD FOR DETERMINING NIGHT BLINDNESS. E. E. METCALFE, U. S. Nav. M. Bull. **38:** 231 (April) 1940.

After exposure to intense white light a lag occurs in correct color appreciation. Green, the last color to recover, is perceived in turn as white, gray-white, blue and finally olive green. The completeness of recovery can be checked with a colorimeter, standard green solutions being used for comparison. In Metcalfe's procedure a lamp that gives 250 foot candles at 5 cm. is focused on the eye for one minute. The examinee then views a white screen 20 feet (6 meters) distant, on which are projected various colors. The recovery time for green is noted. A physiologic variation occurs with age, from about two minutes in the twenties to six minutes in the fifties. In cases of night blindness the recovery time is markedly prolonged.

J. E. LEBENSOHN.

Neurology

EXPERIMENTAL STUDIES ON HEADACHE: FURTHER ANALYSIS OF THE MECHANISM OF HEADACHE IN MIGRAINE, HYPERTENSION AND FEVER. A. M. SUTHERLAND and H. G. WOLFF, Arch. Neurol. & Psychiat. **44:** 929 (Nov.) 1940.

Previously the authors have indicated that the dilatation and distention of cranial arteries form the basis of the headache produced by a variety of factors. The purpose of this communication is to analyze further the mechanism of such headache and to ascertain which cranial arteries are responsible for the headache in each instance.

For example, faradic stimulation of the proximal few centimeters of the anterior middle and posterior cerebral arteries and of the first few centimeters of the intracranial portion of the internal carotid artery causes pain within, behind or over the homolateral eye. Furthermore, stimulation of the vertebral and basilar arteries and the proximal portions of the branches causes pain in the occipital and the suboccipital region. These areas are commonly involved in attacks of migraine.

To correlate the data on the various headaches under consideration, a comparison of the prototypes of each class of headache was made. Histamine headache was taken as the representative of a class which includes the headaches induced by amyl nitrite and foreign proteins and those associated with fever. The migraine headache was taken as representative of a class which includes, as well as the many varieties of migraine, the headache associated with hypertension.

After analyzing these various types of headache from the point of view of production, natural and experimental, and of alleviation, as by vasoconstrictor drugs and manual compression of vessels, they draw the following conclusions. For the histamine headache it is likely that the cerebral branches of the internal carotid, basilar and vertebral arteries at the base of the brain are primarily responsible. To the migraine headache, however, the extracranial, and possibly the dural, branches of the external carotid artery are the chief contributors. R. IRVINE.

Operations

EVISCERATION OF THE GLOBE WITH SCLERAL IMPLANT AND PRESERVATION OF THE CORNEA. F. E. BURCH, Am. J. Ophth. **23:** 47 (Jan.) 1940.

Burch gives the following summary:

"The cosmetic result and general comfort following evisceration without keratectomy exceed those obtained by the method of Mules. Irritation, lacrimation and accumulation of secretion are less than after other methods. A sunken globe or loss of the lid fold does not occur, and the appearance and motility usually defy recognition of the fact that a prosthesis is worn. Patients are pleased with the better cosmetic results obtained. There is relatively less reaction with the technique described than after Mules's operation, involving sacrifice of the cornea. In Mules's operation this sacrifice is unnecessary and possesses no advantage. The results in the series reported have been so satisfactory that further experience with the method described is warranted."

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

TREATMENT OF EXOPHTHALMOS BY DECOMPRESSION OF THE ORBIT: REPORT OF TWO CASES. A. MCCRAVEY and H. R. MATHER JR., Arch. Neurol. & Psychiat. **43:** 187 (Jan.) 1940.

The first case illustrates the danger of delay in decompression of the orbit in cases of progressive exophthalmos. Early decompression

of the orbits would have preserved useful vision in both eyes. In the second case, in which the exophthalmos measured the same in both eyes, a different operative approach was used for each eye. This afforded an excellent opportunity to observe the relative merits of the two procedures. The case is the first to be reported in which treatment has been carried out in this manner. The results to date have been equally satisfactory. However, the Swift operation was less formidable, the operating time being about half that of the Naffziger operation, and convalescence was shorter.

It was interesting to note that in both cases the exophthalmos progressed rapidly after thyroidectomy; however, the toxic signs disappeared.

The extraocular muscles in both cases showed extreme hypertrophy, as has been described by others.

R. IRVINE.

Parasites

OPHTHALMOMYIASIS INTERNA ANTERIOR. C. S. O'BRIEN and J. H. ALLEN, Am. J. Ophth. **22**: 996 (Sept.) 1939.

O'Brien and Allen report the case of a boy who ran a nail into his foot. Ten days later the right eye became red, congested and painful; the lens was found to be subluxated and the tension was 65 mm. (Gradle-Schiötz). Biomicroscopic study revealed a gray, translucent, cigar-shaped object 0.5 mm. in its greatest diameter and 2.5 mm. long in the anterior chamber. A diagnosis of invasion by a larva was confirmed on removal. The larva proved to be Hypoderma. The eye recovered.

W. S. REESE.

Pharmacology

PRESSURE-LOWERING ACTION OF PILOCARPINE AND PHYSOSTIGMINE. K. HRUBY, Arch. f. Ophth. **141**: 517, 1940.

The various theories which have been advanced to explain the lowering of the intraocular pressure by the local application of pilocarpine and physostigmine are based on one or several of the following mechanisms: (1) the unfolding (increase in surface) of the iris; (2) the improved access to the chamber angle due to the movement of the iris toward the anteroposterior axis of the eye; (3) the effect of the caliber of the intraocular blood vessels, and (4) the contraction of the ciliary body.

The author has reviewed the extensive literature with the object of determining which theory offers explanations for the largest number of pertinent clinical phenomena. He arrives at the conclusion that the tension-lowering action of the miotics is probably due to improved outflow from the anterior segment caused by the contraction of the ciliary body. In a material of 281 cases of primary glaucoma (from the Lindner clinic in Vienna) Hruby found the relative frequency of emmetropia, myopia and hyperopia to be the same as in a nonglaucomatous material. If the patients with glaucoma were divided into groups according to

age it was found that myopia was relatively more frequent in the younger patients and hyperopia in the older ones. From this the author draws the conclusion that glaucoma tends to develop in myopes at an earlier age than in hyperopes. Since most of these patients did not wear glasses for the correction of their ametropia, it is possible that the time of the onset of glaucoma had been influenced by the extent to which they had made use of their ciliary muscles prior to the onset of glaucoma. This observation may therefore speak for the theory that the activity of the ciliary muscle is an important pressure-regulating factor through which the miotics lower the intraocular pressure.

P. C. KRONFELD.

Physiology

SOME OBSERVATIONS ON THE PRESENT POSITION OF OUR KNOWLEDGE OF THE INTRA-OCULAR FLUID. STEWART DUKE-ELDER, J. C. QUILLIAM and HUGH DAVSON, Brit. J. Ophth. **24:** 422 (Sept.) 1940.

The article begins with a critical review of the evidence that has been presented in support of the secretory hypotheses. Then follow the results of comparative chemical analyses of the aqueous humor and the blood serum of cats and the studies on permeability.

If the theory of secretion is to be accepted, the following main points must be investigated. First, a definite glandular activity on the part of a tissue in the eye must be demonstrated, and it must be shown that the intraocular pressure is a definite function of the activity of the cells of this tissue. Second, it must be shown that the secretion, if it occurs, is effective in carrying out the objectives the necessity for which is the basis for the assumption of secretory activity. Third, a negative line of inquiry is to be pursued to investigate whether the distribution of dissolved constituents in the aqueous humor and in the plasma is such as to necessitate the assumption of an accumulating or diluting activity on the part of some hypothetic glands.

The authors reach the following conclusions:

"1. . . . The distribution of ions between the aqueous humour and the blood serum is consistent with the conception of dialysation, and shows deviations from theoretical values less than those obtained in dialysates in other in vivo systems and in artificial dialysates of serum. The distribution of non-electrolytes can provide no competent basis for argument.

"2. . . . The permeability between the serum and the aqueous is comparable in the normal animal, in the surviving profused head, and in the perfused head poisoned with cyanide; in these circumstances the existence of any secretory activity in transit is impossible. The results of the present experiments show no difference in general behaviour between the living and the dead eye, a finding which argues against the existence of secretory activity.

"3. . . . The claims advanced for the existence of irreciprocal permeability between blood and aqueous are qualitatively and quantitatively unfounded.

"4. . . . In general terms we consider that it would be unwise to revive the old theory to the effect that the aqueous humour is a secretion elaborated by specialized cells, and that it would be preferable, and more in consonance with the experimental facts, to regard the aqueous humour primarily as an ultra-filtrate of plasm; but, as a result of metabolic activity occurring in the eye, and perhaps of the interposition of a second membrane (as opposed to a secretory activity of the membrane), to conclude that the concentrations of certain dissolved constituents in the aqueous humor are modified so that the observed concentrations represent a steady state and not the static Donnan equilibrium which one would expect in the absence of such disturbing activity."

W. ZENTMAYER.

Retina and Optic Nerve

RETINITIS STELLATA IN CONNECTION WITH FOCAL INFECTION. BELA BOROS, Brit. J. Ophth. **24:** 137 (March) 1940.

The author reports a case of retinitis stellata occurring in a woman 42 years of age. Vision in the affected right eye was limited to the counting of fingers at 15 meters; that of the left eye was normal. The optic disk was filled with blood, and the contours were indistinct. Small glistening white spots were present along the vessels at the disk, toward the macula. At the macula, there was a star-shaped figure, strikingly regular in design. After removal of "ten bad teeth," vision improved to 6/12. At this time the fundus, however, showed no significant changes. Three months afterward, the vision and the fundus became normal, with the exception of a few tiny white spots.

The author concludes that an etiologic connection exists between the focus and the retinitis stellata, and therefore that the endothelial disturbances (Marx; Hellmut) and spasms in the retinal vessels are caused by microbes or toxins carried from the focus into the circulation. As a consequence of this, a deficiency in nutrition occurs, causing the disease of the retina.

Consequently, when diagnosing retinitis stellata one should pay attention to the role of the teeth.

W. ZENTMAYER.

TUBERCULOUS ETIOLOGY OF RETINAL PERIPHLEBITIS. M. WENDLING, Arch. f. Ophth. **141:** 198, 1939.

The author made a study of 87 patients with retinal periphlebitis (136 eyes) who had been under Werdenberg's care at his sanatorium for ocular tuberculosis (Davos, Switzerland). The aim of the study was to answer the following questions:

1. In what percentage of cases can a relation between intrathoracic tuberculosis and retinal periphlebitis be demonstrated?

2. Does anterior uveitis occur in a significant number of cases of periphlebitis?

3. Do therapeutic successes obtained with antituberculosis treatment permit conclusions with regard to the cause?

The results of the physical examination and of thoracic roentgen examination (with soft rays and an intensifying screen) in 73 cases of periphlebitis were as follows:

1. Active intrathoracic tuberculosis.
   A. Exudative tuberculosis of the glands at the hilus, 22 cases.
      (*a*) With dissemination in the lungs, 17 cases.
      (*b*) Without dissemination in the lungs, 5 cases.
   B. Definite pulmonary tuberculosis, 22 cases.
2. Not definitely active or inactive intrathoracic tuberculosis.
   A. Fibrous changes at the hilus, 16 cases.
   B. Fibrous pulmonary tuberculosis, 13 cases.

Definite active pulmonary or hilar tuberculosis was present in 44, or 60 per cent, of the cases. Residues of pleuritic attacks were common. Among the cases of definite pulmonary tuberculosis were 6 in which cavities were present. In 51 per cent of the cases there was parallelism between the severity of the thoracic disease and that of the ocular disease, and in the others there was antagonism—severe ocular disease associated with slight thoracic abnormalities and vice versa.

In 12 per cent of all cases of periphlebitis anterior uveitis was noted; in 23 per cent there were recent or healed choroiditic lesions.

The treatment consisted of climatotherapy, administration of roborants and measures "directed against the hemorrhages" (no details given). Most of the patients stayed in the sanatorium until the ocular as well as the pulmonary disease was well healed. The visual results were as follows:

On Admission		On Discharge	
Vision	Number of Cases	Vision	Number of Cases
0	7	0	9
1/1,000	5	1/1,000	8
1/200 - 1/10	35	1/200 - 1/6	20
1/3 - 1/4	15	1/4 - 1/3	6
1/3 - 2-3	26	1/3 - 2/3	23
5/6 - 1	27	5/6 - 1	49

These favorable results obtained with therapeutic measures directed against tuberculosis support the concept of the tuberculous causation of periphlebitis retinalis.

P. C. KRONFELD.

SO-CALLED LACTATION NEURITIS: REPORT OF CASES. M. GLEES, Klin. Monatsbl. f. Augenh. **103:** 615 (Dec.) 1939.

Lactation neuritis is characterized by headache, general malaise and reduced vision during confinement or shortly afterward. Neuritis of the optic nerve, peripheral contraction of the field of vision and central scotoma are present. There are also occasional tenderness of the eyeball and pain on moving the eyes. Recovery begins after a few weeks or months, and vision is regained to a great extent. The prognosis is relatively favorable. The cause is unknown, notwithstanding several hypothetic explanations.

Glees reports 3 cases, in which the patients were women aged 25, 19 and 33 years respectively. Two of the patients were primiparas; the third gave birth to her eighth child. The optic neuritis occurred in these 3 patients during lactation—in the first case eleven weeks after labor and five weeks after weaning; in the second case four weeks after labor, and in the third case six weeks after labor. The patients in the second and third cases were still nursing. Physical, gynecologic and neurologic examinations failed to furnish an explanation for the ocular disease. Vision, which was practically nil in the first 2 patients, returned to normal. The third patient did not regain full vision during the period of observation.

The final stage proved to be simple atrophy of the optic nerve with minor visual disturbance. Two of the patients showed vascular changes and edema of the retina around the disks and along the large vessels.

K. L. STOLL.

Trachoma

ORAL TREATMENT OF TRACHOMA WITH SULFANILAMIDE. F. JASSERON and G. MORARD, Presse méd. **48**: 234, 1940.

Jasseron and Morard call attention to the oral treatment of trachoma with sulfanilamide and point out that in their experience this substance gave the best results in the chemotherapeutic treatment of trachoma. The authors treated nearly 200 patients. Subjective symptoms, such as pain, photophobia and lacrimation, disappeared in forty-eight hours; pannus and infiltration of the cornea were resorbed in eight days; corneal ulcers acquired epithelium in thirty-six hours; cicatrization was accelerated, and clinical recovery took place in three weeks in 75 per cent of the cases. Secondary infections with Weeks's bacillus, diplobacilli of Morax, pneumococci and gonococci were sterilized in four days. The authors recommend for adults 20 Gm. in thirteen days, for children aged from 8 to 12 years 12 Gm. in twelve days, and for children aged from 4 to 8 years 7 Gm. in twelve days, in decreasing doses. Limited local treatment is advised.

J. A. M. A.

SULFANILAMIDE DERIVATIVES IN TREATMENT OF TRACHOMA. L. POLEFF, Presse méd. **48**: 235, 1940.

According to Poleff, instillation of sulfanilamide derivatives into the eye caused conjunctival hyperemia with dilatation of the vessels, while subconjunctival injections were followed by intense hemorrhage at the place of injection. Repeated instillations and subconjunctival injections in 5 cases were not followed by sterilization of the bacterial flora of the conjunctival sac, while silver nitrate and ethoxydiaminoacridine lactate produced rapid diminution of the bacteria and general improvement. Intramuscular injections alternated with oral administrations of sulfanilamide derivatives were followed by a notable decrease of bacteria in the conjunctival sac; however, the final results were not satisfactory. Oral and intramuscular administration of sulfanilamide derivatives together with the usual treatment of trachoma produced satisfactory results in 15 cases of malignant trachoma. Pallor and rapid loss of weight in the course of sulfanilamide therapy are symptoms of intolerance to the drug.

J. A. M. A.

Tumors

DISTANT HEMATOGENOUS METASTASES IN BOTH EYES ASSOCIATED WITH CARCINOMA MAMMAE: REPORT OF CASES. H. R. SCHINZ, Klin. Monatsbl. f. Augenh. **103:** 425 (Oct.-Nov.) 1939.

Schinz reports distant hematogenous ocular metastases of primary carcinoma of the mammary gland in 3 women. They belonged to a group of 536 patients with malignant tumors of the breast, including a few men, encountered among 3,432 patients treated at the Roentgen Institute at Zurich, Switzerland. This type of metastasis was observed with no kind of malignant tumor except primary carcinoma. The ages of the 3 patients were 48, 39 and 40 years, respectively. Hematogenous distant metastases may be considered as late manifestations and as symptoms of general carcinomatosis. They may be more frequent than is generally supposed. In conformity with previous publications, these metastases were located in the choroid and appeared to have been caused by tumor emboli carried through the short posterior ciliary arteries. The rarity of these metastases from the most frequent type of carcinoma, that of the stomach, may be due to the two "filters," the liver and the lung. The choroid appears to be the ocular tissue most favorable for the growth of tumor emboli carried through arteries and arterioles.

K. L. STOLL.

Uvea

ON THE RELATIONSHIP BETWEEN CHRONIC IRIDOCYCLITIS AND TUBERCULOSIS, AND THE APPROPRIATE THERAPY. W. D. WYKEHAM, F. A. JULER and E. R. WILLIAMS, Brit. J. Ophth. **24:** 317 (July) 1940.

The outstanding findings which emerge from the study of these 40 cases is that in a high proportion clinical and roentgen evidence was found suggestive of the presence of tuberculosis elsewhere in the body. The 40 patients had in common a chronic ocular lesion but were otherwise unselected. The fact that 25 of them showed evidence suggestive of tuberculosis on clinical and roentgen examination is therefore beyond the limits of coincidence and strongly supports the view that chronic iridocyclitis is caused by tuberculosis.

Forty cases of chronic iridocyclitis were studied with a view to investigating the importance of tuberculosis in the causation of the condition.

The clinical features, the ophthalmologic features and the roentgen and laboratory data are presented and discussed.

Treatment of the local lesion and of the patient as a whole is discussed in the light of the responses shown to the various methods employed. These results favor a prolonged rest and a sanatorium regimen.

Postmortem examination was not possible in any of the cases, and in only 2 was an eye excised. Blood cultures were not attempted. Clinical and roentgen evidence of tuberculosis elsewhere in the body was present in 25 of the 40 patients; tubercle bacilli were demonstrated in 7, while histologic evidence of undoubted tuberculosis following biopsy was forthcoming in 9 cases.

The Mantoux reaction and its values are discussed at length. In 36 of the 40 cases the Mantoux test was performed, and the result was positive in 30. Analysis of these results in regard to the dilutions employed showed that when evidence of extensive dissemination was present either the reaction was negative or the more concentrated solutions were, as a rule, necessary to give a positive result.

Detailed reports of the entire 40 cases are appended.

The article is illustrated.

W. ZENTMAYER.

DETACHMENT OF THE RETINA IN CASES OF HETEROCHROMIA (E. FUCHS). K. ŠAFÁŘ, Klin. Monatsbl. f. Augenh. **103:** 344 (Sept.) 1939.

Šafář demonstrated 2 cases of heterochromia in which detachment of the retina occurred in the lighter-colored eye. Brief histories of the 2 patients, a man aged 39 and a woman aged 35, are recorded. The author maintains that the occurrence of detachment in the lighter-colored eye was not incidental but was due to an inferiority brought on by cyclitis. Eyes of this type are apt to have also other complications, such as cataracts, opacities of the vitreous, peripheral chorioiditis and, rarely, glaucoma. Šafář found, however, only 1 case of detachment of the retina in the ophthalmoscopic literature; it was published by Botteri in 1930. Discussing Šafář's paper, H. Rieger mentioned detachment of the vitreous body which he observed in a case of heterochromia.

K. L. STOLL.

Therapeutics

A PRELIMINARY NOTE ON THE USE OF RETRO-BULBAR PROCTOCAINE ANESTHESIA FOR THE RELIEF OF INTRACTABLE OCULAR PAIN. L. H. SAVIN and T. M. TYRRELL, Brit. J. Ophth. **24:** 560 (Nov.) 1940.

Proctocaine consists of procaine 1.5 per cent (for immediate anesthesia), butyl paraaminobenzoate 6 per cent (for prolonged anesthesia after the effect of the procaine has worn off) and butyl alcohol 5 per cent (slightly anesthetic and to assist solution) in almond oil.

The authors claim that in many cases this solution, injected behind the painful eye, gives dramatic and prolonged relief, allowing respite from pain while the eye itself is treated by ordinary therapeutic measures.

The injection is given by ordinary technic for retrobulbar injections, about 1 to 1.5 cc. of the anesthetic being used. Relief from pain should follow the injection and should last up to three weeks. Care should be taken when corneal sensitivity is lowered. Diplopia may occur. No toxic effects have been noted.

W. ZENTMAYER.

VITAMIN B_1 IN HYPOVITAMINOSIS. I. CZUKRASZ, Klin. Monatsbl. f. Augenh. **103:** 221 (Aug.) 1939.

The results of administration of vitamin B_1 in the form of salves or hypodermic injections to 10 undernourished patients are reported. The eyes presented herpes dendriticus, herpes zoster and ulcerating

corneal processes. The salves produced better results than did the parenteral treatment. In the author's opinion the effect was less favorable with chronic conditions due to destruction of the nerve fibers.

K. L. STOLL.

SUCCESSES AND FAILURES OF ROENTGEN IRRADIATION: REPORT OF CASES. A. BADER, Klin. Monatsbl. f. Augenh. **103:** 503 (Oct.-Nov.) 1939.

The author reports 2 cases of treatment with roentgen irradiation —1 case of suprasellar tumor and 1 of a post-traumatic cyst of the iris in the anterior chamber. Swift and apparently permanent cure was obtained in each case. When the suprasellar tumor recurred, after five years, and the cyst of the iris, after four years, irradiation failed in each case.

K. L. STOLL.

LOCAL APPLICATION OF VITAMINS IN DISEASES OF THE EYE. V. A. TIKHOVA, Vestnik oftal. **14:** 16, 1939.

The therapeutic effect of local application of vitamins was observed in 46 patients with ulcers of the cornea, phlyctenular keratoconjunctivitis, trachomatous xerophthalmia, postoperative keratitis and (in 2 cases) postoperative conditions in which restoration of the conjunctival sac was necessary. Cod liver oil (several daily instillations into the conjunctival sac) was used because of its bactericidal qualities and its content of vitamins A and D. Routine therapy had been used previously for these patients but had given no effective results. The majority of the patients noted improvement of photophobia and blepharospasm after a few days of treatment with cod liver oil. The ulcers and infiltrates epithelized quickly; the time of healing was shortened considerably, and the corneal opacities were fine. O. SITCHEVSKA.

Society Transactions

EDITED BY W. L. BENEDICT

BUFFALO OPHTHALMOLOGIC CLUB

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Feb. 13, 1941

A Few Facts About Retinal Detachment. DR. IVAN J. KOENIG.

Prior to 1930, 20 per cent of all the adult blind lost their vision through retinal detachment. Sir William Lister in 1927 expressed a universal opinion when he stated that "to operate on a case of retinal detachment is practically valueless."

The first retinal tear was noted by Coccius in 1853, and a little later one was reported by von Graefe. DeWecker in 1870 thought the tear was the cause of the detachment. An operative procedure of the cautery type was first attempted by Martin, DeWecker and de Luca in 1881. Deutchman in 1896 used ignipuncture at the site of the retinal tear. After this Gonin carried on and correlated the facts presented by others into a principle. Gonin maintained that the hole or retinal tear was the cause of the detachment and laid down the following principles based on anatomic facts: 1. One or more retinal tears are present in almost all cases of idiopathic detachment. 2. Tears are caused by dragging of the vitreous at the site of a previous vitreous-retinal adhesion (described by Vogt). 3. The hole allows the fluid of the vitreous to enter behind the retina, separating it from its pigment layer (Leber's theory). Von Graefe, Sourdille and others have brought about retinal detachment experimentally by inflammatory reaction. However, an exudative process would seem to be the cause only when some element is present to produce it, such as toxemia of pregnancy.

From the time of his first paper on the cure of retinal detachment by the thermocautery method, Gonin continued working and experimenting until the publication of his results in 1929, and since that time his work has been recognized.

Weve, of Amsterdam, modified Gonin's operation by using microcoagulation through the sclera and choroid with a current as low as 50 milliamperes. He placed multiple punctures around the hole, thus sealing it off. Šafář, of Vienna, and Walker, of the United States, popularized the micropin method.

The chemical cautery method of the Guist and Lindner modification should be mentioned; also, the use of the pyrometric electrode of Coppez

with diathermy, the flat coagulation of the sclera by Larssen, the thermophore method of Langdon, and electrolysis, as employed by Vogt, von Szily and Machemer.

Anatomically it is known that detachment develops between the outermost limits of the layer of rods and cones and the epithelial layer of the retina (i. e., in the layer of pigment epithelium). The hole or tear involves the retina down to the pigment epithelium. The hole or tear assumes various shapes and forms; 10 per cent of these are really disinsertions of the retina or a tearing away from the ora serrata and probably a result of cystic degeneration at this point. Tears are seen more easily when they are recent and the vitreous is clear.

The types of electrodes are many. All of them endeavor to create an inflammatory reaction of the choroid to bring about an adhesive choroiditis with the final result of an adhesion between the retina and the choroid.

I use the diathermy micropin coagulation method. During the past four years I have used a single electrode with a micropin at its end. It is easier to manipulate, and the depth of the puncture can be regulated, I try not to penetrate beyond the choroid. With this method it is usually possible to finish without any marked hypotony. I coagulate the entire area of detachment, paying particular attention to the area of tears or disinsertions.

The final results of surgical reattachment give a universal record of about 50 per cent cures. Most of the unsuccessful results are found in cases of long standing. It seems that the longer the surgical treatment is delayed, the greater is the detachment and the less chance there is of restoring visual acuity. These facts emphasize the necessity for early surgical therapy.

The following results of surgical treatment were obtained in a group of 20 cases: successful reattachment in 14 cases and failure in 6 cases. The following observations were made at operation: tears, 12 cases; disinsertions, 3 cases, and no holes, 5 cases.

The following determinations of vision were obtained: 6/45 to 6/30, 3 cases; 6/20 to 6/15, 2 cases; 6/12, 5 cases; 6/9, 2 cases, and 6/6, 1 case.

(A moving picture in color of Dr. Koenig's detachment operation was shown.)

DISCUSSION

Dr. Joseph Schutz: The mechanism of detachment of the retina is thought to be through distention, depression, retraction or exudation. The chief factors in its causation are myopia, trauma, chorioretinitis, senile degeneration of the retina and cystoid degeneration of the retina at the ora. Sixty-seven per cent of the patients with this condition are males and 33 per cent females. It is more common in older persons although in younger ones high myopia or trauma may be a contributing cause. It is necessary to differentiate between serous and flat detachment. If hypotony is present the prognosis is bad. Possible complications of the surgical treatment are iridocyclitis, cataract and secondary glaucoma.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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March 20, 1941

Melanoma of the Conjunctiva with Orbital Involvement: Report of a Case. DR. W. E. FRY.

Not infrequently a nevus, either pigmented or unpigmented, is seen in the bulbar conjunctiva. Most frequently the growth is seen near the limbus, in the region exposed by the palpebral fissure. In a certain number of cases, in a person over 30 years of age such a nevus increases in size either spontaneously or as a result of the irritation of trauma or inflammation and takes on the characteristics of a malignant tumor. The case to be reported is an instance of this occurrence.

M. S., a woman aged 50, was first seen at the ophthalmologic dispensary of the Hospital of the University of Pennsylvania on April 26, 1939. At that time the vision in the right eye was 6/15 and in the left eye 6/15 — 1. The patient had noticed a dark spot on the sclera of the left eye to the nasal side of the cornea since the age of 15 years. No change or difficulty with this was noted until December 1938. At that time there was some bleeding from the left eye, and after irrigation of the eyes with boric acid solution a piece of hard black tissue about the size of the nail of the little finger dropped from beneath the upper lid. After this the bleeding stopped. She had had no pain. From this time the tumor continued to grow. It was painless. A second hemorrhage from the mass occurred several weeks before her admission to the hospital.

An exenteration of the left orbit was done on May 8. At operation it was found that the tumor mass occupied most of the nasal portion of the orbit. Convalescence was uneventful.

The patient was readmitted to the hospital, and on July 10 a skin graft of the left orbit was done.

The patient has been under observation from time to time since her last operation. There has been no evidence of extension or metastasis.

Microscopic Examination.—The section consisted of sheets and a number of irregularly arranged nests of cells. There was a moderate amount of pigmentation. The cells showed variable amounts of cytoplasm. The nuclei were irregular in size; many were large. The diagnosis was malignant melanoma.

Melanoma of the Eye, Intrabulbar and Epibulbar. LT. COL. J. E. ASH.

This paper is based on 1,600 odd specimens of melanoma collected in the Registry of Ophthalmic Pathology at the Army Medical Museum. The striking facts from the data on 1,500 specimens of malignant melanoma of the uveal tract are the rarity of the tumor in the Negro race, its predominance in the choroid and its occurrence in elderly

persons. Recurrence in orbital tissue is not common, which suggests that postoperative irradiation is unnecessary. The two commonest clinical symptoms are retinal detachment and glaucoma. From the number of small flat melanotic tumors found in eyes removed for other causes, one may conclude that a certain percentage of such tumors remain quiescent.

Melanoma of the iris may be treated more conservatively, either by observation or by iridectomy, but not by irradiation.

One of the most valuable things that have come out of the registry is Callender's classification of melanoma by cell type. More recently, a study has been made to determine whether there is any prognostic significance in the argyrophil fiber content of this tumor. A classification by pigment content of 450 specimens of intraocular malignant melanoma followed five years or more is given.

Epibulbar melanoma is also dealt with, and this also is found to affect the Negro but rarely. It would seem that this type may be treated more conservatively, especially where local complete excision is practicable. Again it is felt that irradiation is contraindicated. It seems that the prognosis is definitely better for a tumor which has invaded the cornea than for one which has extended posteriorly into the subconjunctival and episcleral tissues.

Book Reviews

Practical Orthoptics in the Treatment of Squint. Second Edition. By Keith Lyle and Sylvia Jackson. Price 15s. Pp. 341, with 96 illustrations and 5 plates. London, H. K. Lewis & Co., Ltd., 1940.

The first edition of this work, which appeared in 1937, contained 211 pages and 68 illustrations. Although the present edition is over half again as large, there is no evidence of "padding." The text has both brevity and lucidity, attributes which occur only too rarely in the same book. Since much of the pioneer work on orthoptic training was done at the Royal Westminster Ophthalmic Hospital, it is appropriate that two members of the staff of this hospital should have produced what is, in my opinion, a desirable addition to ophthalmic literature.

After outlining the management of an orthoptic clinic and describing the instruments used (synoptophore, cheiroscope, myoscope, stereoscopes, diploscope and Remy separator), the authors discuss the optical treatment of squint and the use of occlusion. Chapters are devoted to the examination of the patient, the etiology of strabismus, abnormal retinal correspondence, suppression, fusion and stereoscopic vision. The principles of operative treatment are outlined.

The remainder of the book discusses the orthoptic treatment of unilateral and alternating convergent and divergent concomitant squints, squints in adults, heterophoria, paralytic squint and ocular torticollis. Certain standards of cure are stated for each type of case, and indications for operation are outlined for use in cases in which orthoptic training is either ineffective or not indicated.

In the present volume the results of treatment have been placed in an appendix at the end of the book. A brief analysis of 511 cases is given, followed by twelve tables containing detailed information about 161 cases.

Despite its small size, this volume contains much valuable information. The authors are to be congratulated for having lived up to the implications of the title of their book, because it deals primarily with the practical aspects of orthoptic treatment. W. F. DUGGAN.

A Textbook of Ophthalmology. By Sanford R. Gifford. Second edition. Price $4. Pp. 470, with 230 illustrations, including 15 in color. Philadelphia: W. B. Saunders Company, 1941.

The first edition of this textbook was reviewed in the ARCHIVES (**20:** 163 [July] 1938). In the second edition the subject matter has been brought up to date with the introduction of advances in the field. This has led to a revision of the section on changes in the eyegrounds in cardiovascular disease, and the chapter on the sclera has been rewritten. A number of new therapeutic agents are reported. Some of the black and

white illustrations have been eliminated and others replaced, and there are 15 new colored illustrations. With these additions the textbook has gained in excellence and can all the more be unreservedly recommended.

It has occurred to the reviewer that the author of a popular small textbook is liable to err in trying to make the book too complete; as the book has to be kept down in size, abbreviations result which are of value only to persons who already are conversant with the subject. There can be no doubt of the need for a short book for the medical student and beginner as well as for a larger book like Fuchs's for the specialist. The smaller book should give an introduction to the important basic sciences, anatomy, physiology, optics and neurology, and stress the importance of acquiring an adequate knowledge of the methods of examination. Then, in the clinical part, the more or less common diseases and their treatment should be fully described, while the chapter on operations should be limited to the simplest operative procedures.

ARNOLD KNAPP.

Neuro-Ophthalmology. By R. Lindsay Rea, London. Second edition. Price, $13.50. Pp. 688, with 218 illustrations, including 22 in color. St. Louis: C. V. Mosby Company, 1941.

The first edition of this textbook, which was favorably reviewed in the ARCHIVES **20**:893 [Nov.] 1938), was reprinted in May 1938, and this, the second edition, is a copy of the first, with an appendix, pages 517 to 627, added. The appendix amplifies subjects previously treated in the text and brings some new matter. The following subjects are taken up: the pupil and its reactions, the muscles and nerves of the eye and its adnexa, papilledema, optic atrophy, pathology of retinitis pigmentosa, cerebroretinal degeneration, angioid streaks, Foville's syndrome, meningioma of the olfactory groove, congenital and degenerative abnormalities, thrombosis of the intracranial sinuses, intracranial aneurysms, tumors of the optic nerve, arachnoiditis, tuberous sclerosis, Sturge's disease, pathologic conditions of the vegetative nervous system and vitamins. The additions are illustrated with three new colored plates and forty new black and white illustrations.

The subject matter of the book has been made much more complete, though it seems a pity that the additions could not have been put in their proper places. That they were not may have been due to the difficulties of publication under present world conditions, but the increase in price from $9 to $13.50 is hardly justified.

ARNOLD KNAPP.

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NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: Atlantic City.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Allen Greenwood, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. John Evans, 23 Schermerhorn St., Brooklyn.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Vaillancourt, 46 St. Louis St., Quebec.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. V. May, 1703 Main St., Marinette.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Marinette. Time: November 1941.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Spencer S. Howe, 103 E. Holly St., Bellingham, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 419 Stimson Bldg., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. E. Rideout, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. Kessler, 311 Center Ave., Bay City, Mich.
Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Davis, 1615 Howard St., Omaha.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Franklin P. Maury, Professional Bldg., Tucson, Ariz.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. J. K. Heckert, 103 N. Washington St., Lansing.
Secretary-Treasurer: Dr. W. D. Irwin, 710 Hanselman Bldg., Kalamazoo.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. H. Bailey, 29 S. Oakland Ave., Sharon.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Edward N. DeWitt, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Henry L. Birge, 179 Allyn St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S.E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. A. L. Hammerel, 208 N. Broadway, Billings.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Secretary: Dr. Vanderbilt F. Couch, 105 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. E. Spear, Dickinson.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Roderick Macdonald, Rock Hill.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia Hotel, Columbia. Time: November 1941.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. E. L. Goar, 1300 Walker Ave., Houston.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.
Place: San Antonio. Time: December 1941.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 628 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. George G. Hawkins, Newport News.
Secretary-Treasurer: Dr. Guy Fisher, 3 E. Beverley St., Staunton.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Black, 7 W. Bowery St., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Sanford Gifford, 720 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Wallace W. Ralston, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. John I. Garret, 57 Stokes Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Paul Southgate, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kirt G. Parks, 605 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Harold Mulligan, 1680 N. Vine St., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Leonard C. Whiting, 121 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Algernon Reese, 73 E. 71st St., New York.
Secretary: Dr. Brittain Payne, 896 Madison Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. James W. Smith, 1016–5th Ave., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Harold Gifford, 1620 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Secretary: Dr. Wilfred E. Fry, 1819 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward A. Weisser, 119–5th Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. J. Avery, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William M. James, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. H. Rommel Hildreth, 823 Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fred Boyle, 490 Post St., San Francisco.
Secretary: Dr. Frank Hand, 450 Sutter St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. F. R. Webster, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. John R. Myers, State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. S. H. Patterson, 1251–15th St., Toledo, Ohio.
Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Sterling Bockoven, 1752 Massachusetts Ave. N.W., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.